SURGERY
GYNECOLOGY AND OBSTETRICS
An International Magazine, Published Monthly

VOLUME 75 NOVEMBER, 1942 NUMBER 5

ANALYSIS OF RELATIONSHIP OF SURGERY AND GASTROSCOPY IN 95 CASES OF GASTRIC TUMOR

RUDOLF SCHINDLER, M.D., and PAUL LETENDRE, M.D., Chicago, Illinois

PAPERS dealing with the surgical aspects of gastroscopy have been published by Hertel and Kallius, Henning, Korbsch, Carey, Schindler and Giere (12), Barnett, Benedict, and Schindler (11). In this paper an attempt will be made to determine, by an analysis of 95 cases of gastric tumor in all of which a gastroscopy had been performed before operation, the significance of gastroscopy to the surgeon. In 91 of these cases the tumor was a carcinoma: in 1 case it was a sarcoma, in 2 cases benign mucosal tumors were present, and in 1 case a benign submucosal tumor. The operations performed in the 91 cases of carcinoma were: resection, 64 cases (total gastrectomy in 6 of these); palliative operation of some kind, 7 cases; exploratory laparotomy, 20 cases. In the sarcoma case an exploratory laparotomy was carried out with biopsy; at a second operation resection with extirpation of the spleen and the tail of the pancreas. The benign mucosal tumors, 2 cases, were excised, and a resection was done in the 1 case of benign submucosal tumor.

GASTROSCOPIC FAILURES

In 5 of the 91 carcinomas the lesion was not seen gastroscopically (Table VI). In all these 5 cases it was known before the gastroscopic examination that a carcinoma was present. Nevertheless, in 3 of these cases the tumor remained hidden from view because of its location in the so called "blind area" of the lesser curvature of the antrum. In fact, there exist gastroscopically three well known "blind areas," areas which because of anatomic reasons cannot be seen at gastroscopic examination, namely: (1) the lesser curvature of the antrum, as mentioned before; (2) a small strip of the posterior wall on which the objective of the gastroscope is sliding and from which it can not be abducted sufficiently to get a picture; and (3) the lower pole of the stomach. No tumors of the last two "blind areas" were overlooked. The other 2 carcinomas, not seen, surrounded and infiltrated the pyloric ring. At gastroscopy the antrum was well observed and peristaltic waves were seen migrating toward the pyloric end of the antrum, finally closing its lumen completely, thus giving the impression of a true pylorus formation. However, comparison with the x-ray findings and later with the specimen showed that this closing did not take place at the level of the true pylorus but proximally to it thereby hiding the pyloric tumor. The fact that a pyloric carcinoma may escape view at gastroscopy, although an apparent pylorus is seen, should be borne in mind. Most pyloric

From the Frank Billings Medical Clinic, Department of Medicine, the University of Chicago.

TABLE I.—CORRECT GASTROSCOPIC AND X-RAY DIAGNOSIS[1]

Patient Unit No.	Gastroscopic impression	X-ray impression	Surgical or pathological diagnosis
1. C. L. 264235	Large carcinoma along lesser curvature from antrum to cardia	Cannot rule out possibility of small neoplasm high up on lesser curvature of stomach; to repeat examination	Large carcinoma infiltrating stomach along lesser curvature and cardia
2. W. C. 223931	Ulcerated carcinoma in antrum and along lesser curvature of stomach	Gastric carcinoma	Gastric carcinoma adherent to pancreas
3. M. M. 250121	Carcinoma of fornix	Carcinoma medial portion cardiac end of stomach	Gastric carcinoma: metastases to diaphragm
4. M. M. 251037	Ulcerated carcinoma of antrum	Large ulcerating carcinoma of the stomach	Gastric carcinoma: metastases to liver
5. A. W. 273781	Extensive carcinoma lesser curvature and anterior wall of stomach	Carcinoma of stomach	Extensive gastric carcinoma
6. C. S. 227748	Infiltrating carcinoma of antrum	Gastric carcinoma	Gastric carcinoma
7. E. E. 221776	Huge ulcerated carcinoma of antrum	Large carcinoma of antrum	Carcinoma distal part of stomach
8. L. B. 233342	Carcinoma pyloric ring stomach	Polypoid infiltrating type of carcinoma distal portion antrum	Papillary carcinoma pylorus
9. S. L. 209547	Huge protrusion anterior wall. Picture suggestive of a gastric carcinoma	Enormous penetrating gastric ulcer. Malignant? Benign?	Carcinoma protruding from lesser curvature and posterior wall of stomach
10. C. W. 211728	Impossible to inflate air, it comes right back. Such a thing is only seen in cases of leather-bottle stomach (linitis plastica)	Scirrhous carcinoma of stomach	Gastric carcinoma with metastases to liver
11. J. M. 212193	Infiltrating carcinoma near angulus	Organic lesion near pylorus: small neoplasm	Small carcinoma pyloric portion of stomach
12. G. K. 214007	Cherry-sized tumor of pylorus most likely carcinoma of pyloric canal	Large ulcerating lesion antrum probably malignant	Circumscribed carcinoma distal end of stomach
13. H. G. 229087	Huge ulcerated carcinoma extending from anterior wall up to cardia	Polypoid carcinoma involving antrum and body of stomach	Carcinoma involving nearly whole stomach
14. J. U. 231906	Ulcerated carcinoma angulus	Large infiltrating carcinoma of lower half of stomach	Carcinoma along lesser curvature of stomach
15. E. B. 232937	Carcinoma lesser curvature stomach immediately below cardia	Carcinoma cardiac end of stomach	Large carcinoma of lesser curvature of stomach
16. W. L. 239779	Large infiltrating carcinoma involving entire stomach	Large carcinoma body of stomach	Large gastric carcinoma
17. J. J. 244180	Huge necrotic carcinoma along greater curvature stomach	Defect along greater curvature stomach. Carcinoma?*	Gastric carcinoma adherent to pancreas
18. O. T. 250416	Gastric carcinoma protruding from lesser curvature behind angulus	Carcinoma of antrum	Carcinoma lesser curvature of stomach: retroperitoneal metastases
19. L. D. 251054	Huge carcinoma posterior wall stomach	Ulcerating carcinoma of stomach with complete obstruction	Gastric carcinoma; metastases in liver
20. D. Z. 251913	Carcinoma lesser curvature and posterior wall stomach	Large polypoid carcinoma body of stomach	Huge gastric carcinoma
21. C. W. 256981	Carcinoma infiltrating whole stomach	Carcinoma midportion stomach	Gastric carcinoma; peritoneal metastases
22. J. R. 261257	Ulcerating polypoid carcinoma posterior wall and greater curvature of stomach	Carcinoma posterior wall and greater curvature stomach	Gastric carcinoma; metastases in liver
23. A. K. *	Carcinoma antrum of stomach	Carcinoma involving whole stomach*	Carcinoma involving great part stomach
24. J. K. 30273	Infiltrating carcinoma greater curvature stomach	Scirrhous carcinoma greater curvature stomach	Gastric carcinoma with lymph nodes metastases
25. G. S. 228329	Carcinoma antrum stomach	Obstructing carcinoma of antrum	Carcinoma distal part stomach
26. J. W. 242765	Carcinoma greater curvature stomach	Large ulcerating carcinoma of antrum	Carcinoma involving antrum stomach
27. S. Y. 199644	Large ulcerated carcinoma	Carcinoma at the angle of stomach	Carcinoma lesser curvature stomach
28. A. B. 221271	Huge ulcerated carcinoma	Gastric carcinoma	Huge cauliflower-like, papillomatous carcinoma stomach
29. S. M. 207472	Extensive infiltrating ulcerated carcinoma anterior wall stomach	Carcinoma lesser curvature stomach	Carcinoma of stomach; metastases to liver

TABLE I.—CORRECT GASTROSCOPIC AND X-RAY DIAGNOSIS[1]—Continued

Patient Unit No.	Gastroscopic impression	X-ray impression	Surgical or pathological diagnosis
30. A. C. 264626	Huge carcinoma involving antrum and lower half lesser curvature stomach	Large soft carcinoma pyloric half of stomach	Carcinoma involving entire lesser curvature of stomach
31. J. D. 264993	Carcinoma of the antrum	Large carcinoma involving antrum and pyloric end of stomach	Large carcinoma distal end of stomach
32. R. H. 7632	Huge ulcerated carcinoma anterior wall of stomach	Carcinoma of the body of stomach	Large carcinoma upper anterior wall stomach
33. J. G. 133606	Well limited carcinoma of the pylorus	Neoplastic involvement of the pylorus	Carcinoma involving the pylorus
34. C. B. 123321	Carcinoma infiltrating antrum, angulus, lesser curvature and posterior wall of stomach	Large carcinoma posterior wall of stomach	Large gastric carcinoma, invasion of gastrohepatic omentum
35. T. Y. 251546	Ulcerating carcinoma of lesser and greater curvature of antrum	Infiltrating, ulcerating carcinoma antrum and half lesser curvature of stomach	Carcinoma lesser curvature of stomach
36. A. B. 201562	Carcinoma upper portion stomach involving the cardia	Diffuse infiltrating carcinoma upper two-thirds of stomach	Carcinoma proximal part of stomach
37. S. C. 152133	Diffusely infiltrating carcinoma of almost entire stomach	Carcinoma involving middle half of stomach	Extensive infiltrating carcinoma of the stomach
38. B. F. 163834	Ulcerated carcinoma antrum and lesser curvature stomach	Ulcerating carcinoma antrum and lesser curvature stomach	Carcinoma distal part of stomach
39. S. S. 182086	Large ulcerated carcinoma	Ulcerating carcinoma angulus and antrum of stomach	Large ulcerated carcinoma
40. J. K. 182968	Infiltrating, ulcerated carcinoma greater curvature and posterior wall stomach	Large ulcerating carcinoma central portion of stomach	Large carcinoma of stomach
41. E. J. 188610	Carcinoma greater curvature antrum stomach	Filling defect greater curvature stomach*	Gastric carcinoma
42. N. K. 190348	Carcinoma lesser curvature of antrum	Carcinoma antrum stomach	Carcinoma distal part of stomach
43. L. B. 198468	Large carcinoma pyloric canal	Carcinoma pyloric end of stomach	Gastric carcinoma adherent to liver
44. J. L. 271722	Carcinoma of pylorus	Diffuse infiltrating carcinoma of gastric antrum	Gastric carcinoma
45. J. N. 206300	Infiltrating carcinoma of upper half of stomach	Medullar carcinoma of stomach	Gastric carcinoma; hepatic metastases
46. H. R. 131178	Extensive carcinoma infiltrating lesser curvature stomach	Obstruction of pylorus, presumably a carcinoma	Gastric carcinoma lesser curvature
47. M. F. 157274	Annular infiltrating carcinoma of antrum	Carcinoma pyloric end of stomach	Carcinoma of antrum
48. J. P. 116473	Ulcerated carcinoma greater curvature of antrum	Annular carcinoma of antrum	Carcinoma distal end stomach
49. J. H. 188632	Large carcinoma lesser curvature extending anterior wall and posterior wall of stomach	Large ulcerated carcinoma lesser curvature stomach	Large carcinoma lesser curvature of stomach
50. S. H. 241004	Large infiltrating carcinoma of stomach	Ulcerating carcinoma of body of stomach	Carcinoma posterior wall stomach
51. E. F. 243384	Distention of stomach by air impossible. Typical of leather-bottle stomach; linitis plastica	Large scirrhous carcinoma of stomach	Large carcinoma involving whole stomach
52. H. Z. 240390	Large carcinoma involving distal part of stomach	Annular carcinoma of antrum	Carcinoma distal end of stomach
53. J. U. 272458	Gastric carcinoma near cardia. Extensive polyposis of stomach	Carcinoma near cardia with multiple polyps in stomach	Local resection of a carcinoma near the cardia. Polyps not found by surgeon. Biopsy of gastric wall taken; thought to be a hypertrophic gastritis. Microscopic diagnosis of biopsy: benign adenomas
54. A. S. 203362	Huge ulcerated carcinoma posterior wall and lesser curvature of stomach	Large gastric carcinoma. Meniscus sign	Gastric carcinoma posterior wall near lesser curvature
55. E. F. 173735	Carcinoma involving almost entire stomach	Large gastric carcinoma*	Large carcinoma infiltrating nearly entire stomach
56. E. C. 179788	Organic resistance in upper part of stomach. Examination impossible; probably a carcinomatous infiltration	Scirrhous carcinoma of stomach	Carcinoma infiltrating entire stomach

[1]Only the first gastroscopic and x-ray examination are considered in every case.
*Indicates an outside case or outside examination.

TABLE II.—TUMORS OTHER THAN CARCINOMA[1]

Patient Unit No.	Gastroscopic impression	X-ray impression	Surgical or pathological diagnosis
1. M. D. 52454	Well circumscribed polypoid carcinoma posterior wall	Tumor along lesser curvature stomach probably carcinoma	Benign adenoma
2. H. B. 229508	Tumor forming gastritis of posterior wall of stomach? Or an infiltrating carcinoma	Indefinite filling defect upper lesser curvature stomach*	Hyperplastic polyps not felt by surgeon at palpation of stomach. Excision of polyps after opening of stomach
3. L. Q. 251259	Polyposis of stomach	Large neoplastic masses in stomach	Leiomyosarcoma of stomach
4. M. S. *	Submucosal tumor, probably a myoma of stomach wall	Gastric carcinoma*	Myxofibroma of stomach

[1]Only the first gastroscopic and x-ray examination are considered in every case.
*Indicates an outside case or outside examination.

carcinomas are so easily and beautifully visualized that the gastroscopist is tempted to exclude the possibility of a pyloric carcinoma as soon as he has seen the contracting pylorus. This obviously is not permissible.

In 5 cases of carcinoma a wrong diagnosis was made (Tables III and V). Retrospectively, it may be said that in at least 2 of them the correct diagnosis would have been possible under consideration of all findings. In 2 cases a benign ulcer was diagnosed, and it seems to us remarkable that this number is so small. An extensive discussion of these cases will be found elsewhere (11). In 1 of these cases 2 ulcers were present and only the upper larger one was seen at gastroscopy; the lower one remained hidden behind a callous wall. This lower one, however, proved to be microscopically a carcinoma; the upper one looked in many sections like a benign ulcer, although in some of them strands of carcinomatous tissue were found. In the second case location of the ulcer in the antrum close to the greater curvature, bleeding of its edge during the examination, and the finding of a second small ulceration should have been interpreted in favor of a malignant growth.

In 2 cases, gastritis was wrongly diagnosed. In the paper in which the cases of tumor forming gastritis have been described these cases have not been mentioned, but it should be realized that not only a gastritis can be mistaken for a carcinoma, but that a carcinoma can be mistaken for a gastritis. This mistake seems to occur infrequently. In the first of these cases the gastroscopic protocol read:

"Half of the pylorus was observed, but no ulcer was seen. There was whitish adherent mucus on the greater curvature of the antrum. The greater curvature of the body showed no marked changes. In the lower portions there were some greyish white depressed spots surrounded by red halos. In the upper portions adherent mucus and large dark red spots were observed."

Accordingly a severe superficial gastritis of the greater curvature was diagnosed but 5 months later an inoperable carcinoma was found at laparotomy. In the second case the gastroscopic observation should have led to the correct diagnosis.

At x-ray examination a crater of the lesser curvature had been suspected, but no ulcer was found at gastroscopy. However, a protrusion of the posterior wall was seen, described, and pictured, which looked soft and velvety and bled during the examination. The diagnosis of a localized hypertrophic gastritis of the posterior wall was made because of the soft appearance, but the bleeding which occurred during the examination should have cautioned us. At laparotomy a carcinoma was found and removed.

TABLE III.—INDEFINITE OR INCORRECT GASTROSCOPIC DIAGNOSIS; CORRECT X-RAY DIAGNOSIS[1]

Patient Unit No.	Gastroscopic impression	X-ray impression	Surgical or pathological diagnosis
1. C. P. 219651	Gastric ulcer benign? Malignant? 50% 50%	Ulcerating carcinoma lower portions stomach	Carcinoma stomach: metastases in spleen
2. F. P. 235720	Huge ulcer crater high lesser curvature believed to be benign	Large crater on lesser curvature probably carcinoma	Carcinoma upper lesser curvature of stomach

[1]Only the first gastroscopic and x-ray examination are considered in every case.

Finally a case was observed in which a year ago a carcinoma had been removed by resection. Invagination of intestinal mucosa into the stomach hid the recurrent tumor.

Thus, in 5 of 91 cases of gastric carcinoma the lesion was not seen and in 5 a lesion was seen but a wrong diagnosis was made.

The 1 case of sarcoma of the stomach (Table II, Case 3) was wrongly diagnosed as polyposis but retrospective analysis of the gastroscopic protocol suggests that this mistake might have been avoided. Because of the rarity of the case and its practical significance the protocol is given in full here.

"The whole stomach was seen and the picture was one which I have never seen before. The angulus was seen and below it there was a soft protruding mass which filled almost the entire antrum. The mucosa covering it showed many dark creases and folds. A dark hole in its upper portion seemed to be the way to the pylorus. Although this mass was unusually soft I would not be able to exclude a malignant tumor from this picture alone. But then other protrusions were seen—5 along the posterior wall and 3 along the lesser curvature. Those of the posterior wall were large hemispherical protrusions, which, however, sloped gently toward the gastric wall. They all were covered by a smooth and soft mucosa and no stiffness was observed. However, the highest of them, lying in the fornix, had a somewhat different aspect. Its surface was nodular and had a definitely polypoid character. The 3 protrusions of the lesser curvature were smaller, the two lower ones having about the size of a cherry and the highest one having the size of a plum. They were covered by smooth mucosa. Their elevation was partly so that a shadow was cast on the surrounding mucosa but in other portions of the circumference there was a gradual sloping. The uppermost of these 3 formations contained many nodes of different size and if we would have seen this one alone, we would not be able to differentiate between a benign and a malignant adenoma. Its combination with many other tumors speaks for a benign polyposis. The mucosa surrounding the highest of these tumors and of the whole upper third of the stomach was grayish in color with branching blood vessels. It still should be mentioned that in the groove between the highest and the next highest excrescence of the posterior wall there was a dark red small depression which might or might not be a small ulceration."

It is obvious from this description that no simple polyposis should have been diagnosed. The aspect was not that of a carcinoma, however, and the gastroscopist probably did not think of the possibility of a sarcoma. The

Fig. 1. Gross specimen of a leiomyosarcoma of the stomach. The gastroscopic description, as given in text, corresponds exactly with this picture, but at gastroscopy the wrong diagnosis of a polyposis was made.

gross specimen (Fig. 1) shows that every detail of the gastroscopic examination including the ulceration had been reported clearly.[1]

In one of the benign mucosal tumors (Table II, Case 1), a sharply circumscribed (type I) carcinoma was suspected but the microscopic sections showed rather regular adenomatous tissue. Obviously the differential diagnosis between a large benign adenoma and a just beginning adenocarcinoma always will be difficult gastroscopically as well as microscopically. The second case will be described under the next heading. The one submucosal tumor seen was correctly diagnosed as such at gastroscopy because of the characteristic symptom of "bridging folds" which were crossing the recess between the tumor and the adjacent mucosa. This symptom has been described elsewhere. The tumor proved microscopically to be a submucosal myxoma.

In 4 cases an *indefinite diagnosis* was made (Tables III and V).

We shall call a diagnosis an indefinite one in which the gastroscopist refused to commit himself definitely but admitted that he was unable to make a differential diagnosis; when to a definite diagnosis merely a qualifying

[1]The surgeon not experienced in gastroscopy will be surprised about the detail in relief and colors reported in the gastroscopic protocols. It should be re-emphasized that the gastroscopist sees living tissue and that he is able to make out much more detail (for instance at the edge of an ulcerative lesion) than the surgeon or the pathologist can find at the inspection of the excised bloodless specimen. This fact accounts for the relative ease with which the gastroscopist often correctly differentiates benign and malignant ulcers in cases in which the pathologist is unable to make the differential diagnosis without microscopic examination (9).

TABLE IV.—CORRECT GASTROSCOPIC DIAGNOSIS; INDEFINITE OR INCORRECT X-RAY DIAGNOSIS[1]

Patient Unit No.	Gastroscopic impression	X-ray impression	Surgical or pathological diagnosis
1. T. S. 11283	Small infiltrating ulcerated carcinoma of the pylorus	Frank lesion of the pylorus: benign ulcer? Carcinoma?	At operation very small prepyloric ulcer believed to be benign. Microscopic diagnosis: an 8 by 8-mm. carcinoma
2. G. A. 130055	Ulcerated carcinoma lesser curvature stomach	Benign gastric ulcer	Ulcerated gastric carcinoma along lesser curvature
3. G. S. 53880	Extensive carcinoma involving almost entire stomach	Pyloric obstruction most likely peptic ulcer	Carcinoma involving whole stomach
4. W. P. 184930	Diffuse infiltrating, ulcerated carcinoma posterior wall antrum	Gastric ulcers*	Carcinoma involving lower part of stomach
5. I. W. 257958	Carcinoma surrounding distal end of stomach	Marked pyloric obstruction with deformity of duodenal bulb with crater	Carcinoma surrounding distal end of stomach
6. J. B. 127408	Carcinoma of antrum almost completely ulcerated	Obstruction of pylorus benign? Malignant?*	Carcinoma lower part of stomach
7. M. L. 190871	Shallow ulcerated carcinoma with blending edge toward cardia above angulus along lesser curvature stomach	Stenosing lesion of the pyloric end of stomach and of duodenal bulb probably peptic ulcer—cannot rule out carcinoma	Carcinoma lesser curvature of stomach
8. L. K. 246122	Dirty ulcerating carcinoma at angulus infiltrating to cardia	Defect in cardiac end of stomach—may be carcinoma	Carcinoma involving whole stomach and lower part of esophagus
9. S. *	Localized carcinoma of distal portion of the posterior wall close to lesser curvature stomach	Inconstant filling defect along lesser curvature*	Gastric carcinoma; hepatic metastases
10. R. C. 185782	Large ulcerated carcinoma on angulus	Ulcer or possibly medullary carcinoma lesser curvature antrum	Carcinoma involving distal end of stomach
11. J. G. 193877	Carcinoma in pyloric ring	Stenosing lesion in pyloric end of stomach. Opinion on nature of lesion is being deferred	Irregular ulcerating carcinoma in pylorus
12. S. H. 145190	Huge carcinoma lesser curvature antrum and body of stomach	Obstructing lesion at pylorus, neoplasm? Ulcer?	Carcinoma lower half of stomach
13. B. L. 242905	Diffusely infiltrative carcinoma posterior wall of antrum	Organic lesion of some sort greater curvature pyloric antrum, neoplasm? Films negative	Carcinoma of stomach posterior wall near lesser curvature
14. L. L. 114109	Large carcinoma along lesser curvature stomach	Penetrating gastric ulcer, cannot absolutely rule out carcinomatous infiltration	Ulcerated carcinoma lesser curvature stomach
15. J. J. 199313	Huge carcinoma of gastric antrum	Constricting lesion of antrum with possible ulceration. Malignant? Benign?	Carcinoma pyloric end of stomach
16. M. N. 76998	Ulcerating carcinoma lesser curvature of antrum and lower third of lesser curvature stomach	Ulcerating lesion of gastric angle: Benign? Malignant?	Ulcerating carcinoma lesser curvature stomach

[1]Only the first gastroscopic and x-ray examination are considered in every case.
*Indicates an outside case or outside examination.

sentence was added, the diagnoses were not considered to be indefinite. The gastroscopist naturally knows that his method is not foolproof and he can not be blamed therefore if he adds the word "probably" to a definite diagnosis. This, however, occurred very rarely in our material. The gastroscopists tried to be as definite as possible in order to find out the limitations of the method.

The first of the 4 cases with indefinite diagnosis has been reported elsewhere (Table V, Case 4). At x-ray examination an ulcer of the posterior wall had been seen and had been interpreted as a benign ulcer. The gastroscopic protocol read:

". . . . All parts of the mucosa were swollen and stiff. The folds looked caterpillar-like. One fold of the posterior wall was so prominent and thickened that it might have been malignantly infiltrated."

This could not be determined with certainty; however, a short time later the diagnosis of carcinomatous infiltration was made at gastroscopy. The autopsy performed 1½ years later revealed carcinomatous infiltration of the entire stomach. In the second of the cases with indefinite diagnosis (Table V, Case 2) an area of infiltration at the midportion of the posterior wall was seen, the nature of which the gastroscopist was unable to determine. In the third of these cases (Table III, Case 1) the

TABLE V.—INDEFINITE OR INCORRECT GASTROSCOPIC AND X-RAY DIAGNOSIS[1]

Patient Unit No.	Gastroscopic impression	X-ray impression	Surgical or pathological diagnosis
1. C. W. 257337	Stiff wall-like fold along lesser curvature; might be malignant	Normal stomach, peculiar deformity of antrum	Carcinoma of stomach adherent to pancreas
2. R. H. 264987	Area of infiltration midportion of posterior wall; nature?	Normal stomach	Gastric carcinoma; metastases to liver
3. M. M. 233159	Area of mild hypertrophic gastritis of lower posterior wall	No definite pathology	Small carcinoma on posterior wall
4. G. L. 162012	Thickening of infiltrated folds, unusual, inflammatory? Malignant?	Gastric ulcer upper lesser curvature	Gastric carcinoma; peritoneal metastases
5. E. C. 184065	Severe superficial gastritis of greater curvature	Prepyloric ulcer lesser curvature	Carcinoma stomach adherent to pancreas
6. E. B. 249253	Benign ulcer anterior wall of antrum	Hourglass constriction of antrum due to ulcer, probably not gastric carcinoma	Carcinoma pyloric end of stomach
7. M. F. 184065	Gastric ulcer—slight hypertrophic gastritis. Probably invagination of intestinal mucosa rather than tumor	Polypoid mass: lesion or redundant mucosa in region of gastroenterostomy	Recurrent carcinoma at the gastroenterostomy

[1]Only the first gastroscopic and x-ray examination are considered in every case.

gastroscopist saw an ulcer with a wall and stated:

"I have never seen so strikingly prominent a delimiting wall in a benign ulcer. If this wall is due to edema in the edge of the ulcer there is at least a 50 per cent chance of its being benign. A very sharply defined, localized large carcinomatous ulcer might give such an appearance."

Operation revealed just such a sharply limited carcinoma.

The last of these indefinite diagnoses (Table V, Case 1) seems to be especially important. The first gastroscopic protocol read:

"There was a resistance 3 cm. below the cardia and in spite of protracted attempts and two introductions it never became possible to introduce the instrument into the lowest depth of the stomach. The mucosa looked definitely pathological. There was a wall-like fold of the lesser curvature and posterior wall which contained hemorrhages but which did not look quite so stiff and infiltrated as we are accustomed to seeing in malignant neoplasms. The mucosa of the fornix and posterior wall were swollen and the greater curvature was slightly protruded. . . . Impression: Unsatisfactory examination. Obstacle 3 cm. below the cardia; stiff wall-like fold along the lesser curvature which might be the uppermost end of a malignant infiltration; however, I can not commit myself definitely."

The patient was treated medically for 7 months. The operation revealed a carcinoma of the upper portion of the stomach.

Finally a case must be added which cannot be considered as a gastroscopic failure, but rather as an unsatisfactory examination. It was impossible to introduce the gastroscope into the stomach because of a stiff resistance at the cardia, which later proved to be due to a carcinoma. It should be mentioned that the sensation of the experienced gastroscopist when he tries to overcome an organic resistance is quite characteristic. The introducing fingers feel a stiff board-like resistance which is definitely different from the resilient resistance felt sometimes in spasms. However, some experience is needed for the evaluation of this sensation; but after all extensive experience is required for the interpretation of the gastroscopic pictures also.

SUPERIORITY OF GASTROSCOPY TO PALPATION OR INSPECTION

It should not be expected that tumors can be seen at gastroscopy which the surgeon is unable to palpate; nevertheless it does occur. This unexpected superiority of gastroscopy has become apparent in some cases of tumor forming gastritis (8, 10).

Three cases of our series should be quoted in this connection.

In the first instance localized tumor formation was seen in the posterior wall immediately below the cardia (Table II, Case 2). On palpation the surgeon was unable to feel anything. Because of the gastroscopic picture the anterior wall of the stomach was incised. The surgeon introduced his finger into the stomach and then he was able to palpate a number of hard papillomas in exactly the location indicated by the gastroscopist. They were removed bluntly and the mucosa was sutured

TABLE VI.—LESIONS NOT SEEN BY GASTROSCOPY; SEEN BY X-RAY[1]

Patient Unit No.	Gastroscopic impression	X-ray impression	Surgical or pathological diagnosis
1. A. B. 140952	No tumor seen	Small carcinoma lesser curvature pyloric end stomach	Carcinoma antrum of stomach
2. W. M. 248258	No tumor of pylorus; pyloric or prepyloric ulcer unlikely	Constant prepyloric defect, probable carcinoma*	Carcinoma stomach
3. B. L. 271050	Nonvisualization of a tumor of the pylorus	Obstructing carcinoma at the outlet of the stomach	Carcinoma pyloric end of stomach
4. B. M. 269039	Normal stomach	Ulcerating neoplasm involving the antrum	Carcinoma involving gastric antrum
5. C. L. 220919	Tumor not seen lying in blind area of antrum	Ulcer of antrum either benign? Or malignant? Strong possibility of malignancy	Carcinoma lesser curvature of antrum

[1]Only the first gastroscopic and x-ray examination are considered in every case.
*Indicates an outside case or outside examination.

from the inside of the stomach. The growths proved microscopically to be benign hyperplastic polyps.

In the second case—a patient with minor epigastric distress—an ulcer had been seen at x-ray within the pyloric canal (Table IV, Case 1). At gastroscopy an unusually small cancer of the pylorus with concomitant atrophic gastritis was definitely diagnosed. The surgeon was not able to feel much during the operation and for this reason performed a rather limited resection. The microscopic examination revealed an exceedingly small cancer (8 by 8 millimeters with a depth of ½ to 2 millimeters). The patient died later from metastases.

In the third case the discrepancy between palpation and inspection at surgical interference and the gastroscopic findings was especially interesting (Table I, Case 53). At 2 gastroscopies the following was observed:

"The picture seen was unique. I never have seen even an approximately similar picture before. Antrum, angulus, and pylorus were normal, but the entire body presented extensive spectacular changes. Its whole wall was somewhat infiltrated and within this infiltration many polyp-like, round hemispherical pearl-like elevations were seen, partly single, partly in groups like flower beds. (At a gastroscopy carried out after the operation 26 separate adenomas were counted.) Each single node had a normal mucosa and there was no ulceration but the top of each node looked almost white and this white color blended with the surrounding mucosa. In the fornix a huge tumor came into view. . . . Toward the anterior wall its surface looked white and necrotic, toward the posterior wall there was a deep red bleeding ulceration. This tumor looked pretty sharply circumscribed." The gastroscopic impression therefore was: "1) carcinoma of the cardia, 2) extensive polyposis of the body of the stomach."

However, at the operation the surgeon was well able to palpate the circumscribed cancer but he was not able to palpate anything pathological in the body of the stomach beneath this growth. As the neoplasm was sharply defined he decided to make a local excision of the tumor because the general condition of the patient made total gas-

TABLE VII.—LESIONS NOT SEEN BY X-RAY; SEEN BY GASTROSCOPY[1]

Patient Unit No.	Gastroscopic impression	X-ray impression	Surgical or pathological diagnosis
1. D. L. 200796	Gastric carcinoma consisting of 2 ulcerated nodes with wall toward greater curvature	Normal stomach	Ulcerated carcinoma midportion of the stomach
2. J. M. 203949	Large ulcerated carcinoma of antrum no sharp edges	No definite diagnosis made*	Large carcinoma lower part of stomach
3. W. G. 187108	Diffuse infiltrative carcinoma of antrum	No definite pathological findings*	Large carcinoma lower part of stomach
4. E. G. 263965	Carcinoma greater curvature of gastric antrum	Normal stomach	Carcinoma lower part of stomach
5. J. R. 164708	Large deep ulcer upper posterior wall more likely malignancy	Probably normal stomach	Gastric carcinoma high on posterior wall

[1]Only the first gastroscopic and x-ray examination are considered in every case.
*Indicates an outside case or outside examination.

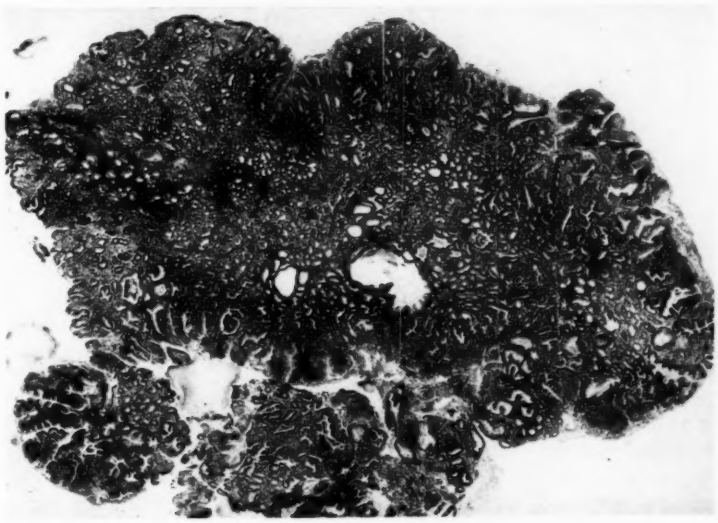


Fig. 2. Benign gastric adenoma in a case of multiple adenomas found together with a gastric carcinoma. In spite of the correct gastroscopic diagnosis the surgeon was not able either to palpate the multiple benign tumors or to see them after the opening of the stomach. The biopsy specimen taken was believed to be a part of a hypertrophied gastric fold. The microscopic picture is that of a typical adenoma. At subsequent gastroscopies 26 such tumors were counted.

trectomy too hazardous. After the stomach had been opened the inside of the body was inspected and only hypertrophied mucosa was seen. A biopsy was taken and this proved microscopically to be a typical adenoma (Fig. 2). How is it possible to explain the discrepancy between the gastroscopic and the surgical inspection? At the surgical interference the stomach was collapsed; at the gastroscopic examination the stomach was distended with air and by this procedure the relief was unfolded, the many polyps becoming easily distinguishable.

COMPARISON OF X-RAY AND GASTROSCOPIC FINDINGS

This paper does not intend to discuss the x-ray aspects of this series. However, the surgeon would like to know whether or not gastroscopy can give him additional information in gastric carcinoma cases not obtained by x-ray. X-ray examination is, and will remain, the standard method for the diagnosis of gastric carcinoma. It has been stated frequently (15) that the close co-operation of x-ray and gastroscopy will give the most ideal results and the series reported will bear out this fact again. Exactly as in the description of gastroscopic failures only the first x-ray examination will be considered. Its result often was altered by repeat examination, frequently undertaken after the first gastroscopic examination. The diagnosis made by the roentgenologist will not be considered as having been indefinite if there was only a minor qualification such as: "probably malignant ulceration" or "benign ulcer but malignant tumor cannot be excluded with the last certainty." Both the gastroscopist and the roentgenologist should make such type of qualification in practically every case since neither method is foolproof. An indefinite diagnosis, however, was assumed if the examiner stated that he was unable to commit himself concerning the differential diagnosis.

Of the 91 gastric carcinomas 8 were not seen at the first x-ray examination (Tables IV, V and VII). In 7 cases a wrong diagnosis was made at the first x-ray examination (benign ulcer); thus, there were 15 failures.

If the diagnoses of both methods are put together, then the total result becomes rather favorable. In 3 cases the diagnosis was not made with either method. These cases require special discussion. In one of them at x-ray some lesion of the lesser curvature was suspected—probably a benign ulcer. At the subsequent gastroscopy this ulcer was not found but a definite bleeding protrusion of the posterior wall was noted but misinterpreted as localized hypertrophic gastritis. Upon reexamining the x-ray films it becomes obvious that under consideration of the gastroscopic findings the diagnosis would have been made.

In the second case x-ray examination revealed an hourglass constriction of the antrum which was due to an ulcer and probably was not cancerous. A benign ulcer, located in the antrum close to the greater curvature, was diagnosed at gastroscopy, too. Both, roentgenologist and gastroscopist agreed after the third and second examinations, respectively, that the lesion might be a cancer, whereupon, 4 weeks after the admission, a resection was carried out. The patient recovered.

In the third case the lesion was not seen by the gastroscopist but on x-ray examination was diagnosed as a benign prepyloric ulcer. This is the only case in which both methods even with our present experience would have led to a wrong diagnosis.

In 3 cases in which the x-ray diagnosis was wrong, the gastroscopic diagnosis was indefinite (suggesting further observation); and in 1 case in which the gastroscopic diagnosis was wrong, the x-ray diagnosis was indefinite (suggesting further observation).

In summarizing we may say that with sufficient experience the close co-operation between roentgenologist and gastroscopist will lead to excellent results in the diagnosis of gastric carcinoma. Neither method is infallible but used together they will result in error in less than 3 per cent and in indefinite diagnoses in only about 4 per cent of cases.

SIZE AND SITE OF TUMOR

If gastroscopic orientation, which requires thorough study, is understood then the location of the tumor thus revealed will not be difficult. In our series the site of the tumor was determined always with the greatest possible accuracy.

How far away from the cardia the upper limit of the tumor is located is often a question of importance to the surgeon. Obviously the gastroscope can answer this question only in so far as the invasion of the mucosa is concerned. The mucosa may not contain any tumor, but the tumor may invade the subserosa and serosa at the same level. Therefore, the gastroscopist usually is careful when he makes the statement that the gastric wall is not invaded between the upper limit of the tumor and the cardia. Often, however, the infiltration of the cardia itself can be definitely noted at gastroscopy. The gastroscopist will also be accurate in his examination of cases in which a wall limits a tumor toward the cardia. The procedure by which the distance of this wall from the cardia is determined is as follows: The gastroscope is withdrawn slowly until the upper limit of the wall crosses exactly the center of the visual field. At this point the distance from the teeth to the rim of the ocular is taken with a tape measure. Then the gastroscope is withdrawn again until the sickle-shaped fold of the cardia crosses the center of the field. The distance, teeth-ocular, is taken once more and subtracted from the first figure. The difference indicates the distance of the wall from the cardia.

The determination of the size of the tumor requires more experience, but with experience can be determined rather accurately. The size seen at gastroscopy depends upon the distance of the object from the objective: if close to the objective the lesion appears magnified; if it is remote it appears diminished in size. The beginner who often does not realize how far away from the objective the respective lesion is will usually underestimate its size. In our series no important mistakes were made in the estimation of the size of tumor; usually the estimation corresponded very closely with the actual size found at operation.

GROSS TYPE OF TUMOR

In recent years it has become customary not to consider all gastric carcinomas as an entity but to classify them according to gross types (3). It has been thought that the final

surgical prognosis might depend upon these gross types (14). Gastroscopically these types can be diagnosed correctly only if the entire circumference of the tumor is seen, or if the diffuse infiltrative nature of the lesion is beyond any doubt. Our experience has taught us to be cautious.

In 17 of 91 cases of gastric carcinoma no gross typing was possible at gastroscopy.

Three cases were believed to belong to Borrmann's type 1. The type 1 carcinoma is the sharply circumscribed polypoid growth. Two of these 3 cases proved to be type 1 carcinomas in the resected specimen, but one of them was a type 2 carcinoma, the wall of which emerging behind the angulus wrongly suggested a polypoid tumor.

Fifteen cases were believed to belong to type 2 at gastroscopy. Borrmann's type 2 is the sharply limited carcinomatous ulcer which often is surrounded by a high wall. This type gives the most colorful gastroscopic pictures and usually can be easily recognized. Fourteen proved to be type 2 carcinomas in the resected specimen and only 1 of them was a diffusely infiltrating type 4 carcinoma.

In 20 cases a type 3 carcinoma was diagnosed. The carcinomatous ulcer of Borrmann's type 3 is rather sharply limited only at one side but it shows diffuse blending and infiltration at the other side. In 19 cases this gastroscopic diagnosis proved to be correct, and in only one case it was wrong. This case, however, seems to us to be important because it later proved to be a type 2 carcinoma.

In 36 cases a diffusely infiltrative type 4 carcinoma was diagnosed but this diagnosis proved to be incorrect in not less than 7 cases. In 3 of these cases a type 3 carcinoma was found in the gross specimen and this mistake does not seem to be a serious one since the prognosis of type 3 and type 4 carcinomas probably is not very different. However, in 4 cases a type 2 carcinoma proved to be present and this is a mistake which should be taken seriously. There is no doubt that the type 2 carcinomas on the whole give a splendid surgical prognosis and certainly it is better not to try typing at all at gastroscopy than to mistake a favorable type 2 carcinoma for an unfavorable type 3 or 4 carcinoma.[1] This experience will caution us to be more careful with the diagnosis of gross types at gastroscopy and to undertake such a diagnosis only if the tumor is seen so completely that no doubt can possibly exist. Since the question of the prognosis of the different types has not yet been settled satisfactorily, the surgical action was not undertaken under consideration of types; all carcinomas were treated in the same way so that no harm was done by the incorrect gastroscopic interpretations.

[1]Thus, in this series type 1 carcinoma was found in 2.7 per cent, type 2 in 27 per cent, type 3 in 29.7 per cent, and type 4 in 40.6 per cent of 74 cases.

SUMMARY

1. In 95 cases of gastric tumor gastroscopic were compared with surgical findings.

In 5 of 91 cases of gastric cancer the lesion was not seen and in 5 other cases the lesions seen were wrongly interpreted. A sarcoma was mistaken for a polyposis, one benign submucosal tumor and one benign mucosal tumor were diagnosed correctly, one benign mucosal tumor was called cancer at gastroscopy. It is believed that the number of gastroscopic failures can be reduced with increasing experience. In 4 cases the gastroscopist did not commit himself as to the nature of the lesion.

2. In 3 cases of this series gastroscopic examination revealed lesions not palpated or seen by the surgeon at laparotomy. Further examination proved lesions to be present.

3. X-ray and gastroscopy will yield the best results in the diagnosis of gastric tumors when used together. In our series of 91 carcinomas 15 failures at the first x-ray examination are compared with 10 failures at the first gastroscopic examination.

4. The size and the site of the tumor were determined satisfactorily by gastroscopy.

5. The gross type of carcinoma should be diagnosed at gastroscopy only if whole circumference of tumor can be seen, and examiner should be careful not to diagnose a diffusely infiltrative type instead of a sharply limited tumor.

REFERENCES

1. Barnett, Crawford F. South. Surgeon, 1937, 6: 502.
2. Benedict, E. B. Am. J. Surg., 1928, 40: 5.
3. Borrmann, R. In Henke, F. & Lubarsch, O. Handbuch der speziellen pathologischen Anatomie und Histologie. Vol. 4, part I, p. 865. Berlin: Julius Springer, 1926.
4. Carey, B. Surg. Gyn. Obst., 1937, 65: 447.
5. Henning, N. Zbl. inn. Med., 1933, 3: 49.
6. Hertel, E., and Kallius, H. U. Arch. klin. Chir., 1928, 151: 578.
7. Korbsch, R. Deut. med. Wschr., 1933, 59: 717.
8. Schindler, R. Am. J. Digest. Dis., 1939, 6: 523.
9. Idem. J. Nat. Cancer Inst., 1941, 1: 451–472.
10. Idem. Chapter (p. 523–541) in F. W. Bancroft, Operative Surgery. New York: Appleton Century, 1941.
11. Schindler, R., and Arndal, O. Arch. Surg., 1942, 44: 473–488.
12. Schindler, R., and Giere, N. Arch. Surg., 1937, 35: 712.
13. Schindler, R., and McGlone, F. Arch. Surg., 1940, 41: 1483.
14. Schindler, R., Steiner, P. E., Smith, W. M., and Dailey, M. E. Surg. Gyn. Obst., 1941, 73: 30–39.
15. Templeton, F., and Schindler, R. Am. J. Roentg., 1939, 41: 354–367.

POSTTRAUMATIC DYSTROPHY OF THE EXTREMITIES

Sudeck's Atrophy

DONALD S. MILLER, M.D., F.A.C.S., and GEZA de TAKATS, M.D., F.A.C.S.,
Chicago, Illinois

IN a previous communication (11) one of us (G. de T.) studied a few cases of vasomotor and trophic disturbances which were interpreted as being due to an abnormal nutritional reflex originating from the site of injury or infection and persisting for months and years. Rightly, this syndrome should be named Sudeck's atrophy, as this author clearly described it in 1900, in 1931, and again in 1938 (35). Since his original description, however, a variety of names has been used to designate the same syndrome such as acute atrophy of bone, traumatic angiospasm, chronic traumatic edema, peripheral trophoneurosis, posttraumatic osteoporosis and reflex nervous dystrophy (11). Obviously, the authors have focused their attention to different manifestations of the same syndrome in the bone, in the vessels, and in the nerves. Its resemblance to causalgia has also been pointed out (11) and recently Homans has reported some instructive cases under the name of minor causalgia. While Sudeck's name is rightfully associated with this condition, he himself preferred to omit his name; atrophy is not a typical feature of the disease in its earlier stages.

In this report we wish to present further clinical data on this vaguely understood and often unrecognized syndrome. A group of clinical cases has been studied with special emphasis on plethysmographic readings giving information on changes in blood flow to such extremities. Finally an interpretation of these findings has been attempted with some hints in regard to the prevention of this disabling clinical entity.[1]

From the Department of Surgery, University of Illinois, College of Medicine and the Fourth Surgical Service, St. Luke's Hospital, Chicago.

[1]Through the courtesy of Drs. Will F. Lyon and Arthur E. Conley we were privileged to study some of the traumatic injuries on their service.

THE INCIDENCE OF POSTTRAUMATIC DYSTROPHY

A vague syndrome such as this is frequently unrecognized, especially in its milder forms. For this reason it is very difficult to find reliable data in the literature on the incidence of posttraumatic dystrophy. Shuele, who has searched a large material for the symptoms and signs of this disease found an incidence of 5 per cent among 500 cases of sprains. Certainly since our attention has been focused on Sudeck's atrophy many cases of minor severity have come to our attention. It is our belief that the early recognition of the peculiar reflex originating from the site of trauma may successfully abort the late sequelae and the long lasting disability by instituting prophylactic and simple therapeutic measures to be discussed below.

THE NATURE OF THE REFLEX

In 1866 Christian Lovén described a reflex, which since bears his name. In stimulating the posterior auricular nerve of the rabbit he found a vasodilatation in the ear on the same side. This in turn could be prevented by cutting the cervical sympathetic trunk. He also found that when a peripheral nerve in the hindleg was stimulated, the volume of the limb increased. Bayliss studied the Lovén reflex and found that the dilatation was due both to the excitation of dilators in the dorsal roots and the inhibition of constrictors in the sympathetic outflow. He published convincing graphs to this effect, some of which were republished in our first communication (11).

A study of these curves reveals, however, that the Lovén reflex, consisting of active vasodilatation and edema of the limb, may be elicited in the absence of the sympathetic innervation. This would indicate that neither

the afferent nor the efferent arc of the reflex need go through the sympathetics. This point must be emphasized because, as will be shown in the study of our clinical cases, increased blood flow, which is one of the cardinal symptoms of the first phase of reflex dystrophy, is not abolished after sympathectomy but persists even though the pain disappears.

Attempts have been made in the past to construct diagrams for such reflexes. Andrews saw the temporary disappearance of edema following radical amputation of the breast, when section of the posterior roots was done for intractable pain. In his diagram the vasomotor reflex arc is led through the sympathetic chain to the posterior root ganglion and from there into the posterior horn; the efferent pathway was led through the anterior roots joining the vessels through the sympathetic nerves. Middleton too believes that there are afferent, sensory fibers in the sympathetics. Yet, until disproved, the experiments of Moore and Singleton have yielded clear cut evidence that painful stimuli from the arteries of the extremities are mediated only by somatic nerves. In man the observations of Foerster and that of Leriche are often quoted to indicate that under a spinal anesthesia, with blocked somatic nerves, pressure or ligature of arteries may still be painful. Repeated personal experience has shown, however, that arterial punctures are painful in the sympathectomized extremity; it is reasonable to assume that vascular sensitivity is mediated by somatic nerves.

It has been common physiological teaching that the stimulation of peripheral nerve is more apt to lead to vasodilatation (i.e., to a Lovén reflex) when the nerve has been cooled previously, when it is in a state of regeneration or when the stimulus is weak. Otherwise, stimulation of sensory nerves usually elicits a pressor effect. Ranson and Billingsley explained the difference in vasomotor responses elicited by weak or strong stimulation by the variations in the resistance to afferent conduction. According to their findings weak impulses reaching the spinal cord are conveyed upward in the depressor path, namely in ventral paths of lateral funiculi containing long fibers with few relays, and result in vasodilatation.

The Lovén reflex then is a form of a defensive phenomenon by means of which an organ or a limb provide themselves with a better blood supply at the expense of the rest of the body. Its afferent arc is no doubt supplied by somatic sensory nerves; but much speculation may arise as to the mode of efferent vasodilatation. The posterior root vasodilators have been called "antidromic" sensory fibers by Bayliss, who believed that they carry impulses in both directions. But evidence has been accumulating that there are vasodilator efferent fibers in the dorsal roots, whose cells lie in the dorsal root ganglions and which are accessible to reflex activation via synapses within the cord (5). Such parasympathetic efferent fibers may truly be the "trophic" nerves of the older authors as their excitation produces vasodilatation and because their excitation mostly occurs as a reflex phenomenon. A herpes zoster is an example of a stimulation of these vasodilator efferent fibers; the stimulation here is due to a vascular or inflammatory lesion of the posterior root ganglion.

Reflex discharges over the dorsal roots were described by Gotch and Horsley in 1891. But their observations passed unheeded because they stood in opposition to the strongly entrenched law of Bell and Magendie. A study of Magendie's article reveals, however, that he did not suggest that sensory, afferent conduction was the only function of the posterior roots. The arguments for and against the existence of posterior root efferent fibers have been reviewed by Hinsey. Recently Toennies has shown conclusively that following the stimulation of sensory nerves in the cat, a reflex discharge takes place over the dorsal roots both on the homolateral and the contralateral extremity. The contralateral reflex is much smaller but lasts longer. A calculation showed that at least 35 per cent of the alpha fibers of the saphenous nerve were accessible to reflex activation.

Important from the standpoint of our clinical problem is the finding that facilitation of the reflex may be observed when a dorsal root to dorsal root reflex was augmented by the simultaneous stimulation of the sciatic nerve.

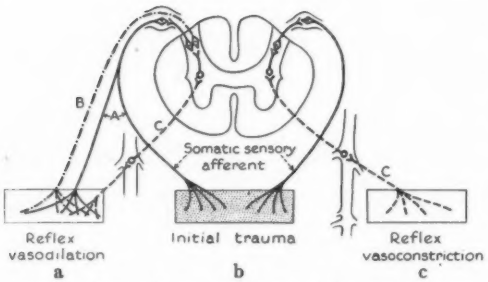


Fig. 1. Trauma in the broad sense of the word may consist of mechanical, chemical, thermal injury, or vascular occlusion. Somatic sensory fibers carry impulses centrally from such an area. In *A*, the nerve impulse never reaches the cord, but produces an efferent vasodilatation, mediated by posterior root efferent dilators (nocifensor nerves). In *B*, the vasodilatation has been activated through a spinal cord reflex. This pathway is postulated by Toennis but its existence is denied by Dole and Morison. In *C*, a vasoconstriction through activation of the efferent sympathetic fibers is shown. Vasodilatation may also occur through the sympathetic outflow. Contrary to the much emphasized reflex vasoconstriction following trauma or thrombosis, reflex vasodilatation is frequent and, if it persists, gives rise to chronic posttraumatic edema, osteoporosis, and burning pain. In carefully conducted animal experiments Albert (*Arch. internat. physiol.*, 1924, 22: 391) showed that all trauma results in vasomotor reactions which are most marked if articular or periarticular structures are injured. In the dog, at least these reactions are predominantly vasodilator, but there is a spinal vasoconstrictor reflex. The vasodilator reflex is abolished only when a peripheral nerve is sectioned and time has been allowed for degeneration. The vasomotor reactions in Sudeck's atrophy are the result of two opposing phenomena, which are vasoconstriction and vasodilation, with the latter predominating at least in the early stages. Sympathetic block abolishes the reflex vasoconstriction and vasodilation through the anterior roots but does not block the axon reflex.

From a series of careful and painstaking clinical observations, Sir Thomas Lewis (23) was also forced to conclude that a set of nerves must exist outside of the somatic and sympathetic fibers which are responsible for a diffuse and widespread hyperalgesia, which appears in many subjects around a point of faradic stimulation or a tiny crushing of the skin. These nerves, he believes, influence cell permeability and cause a release of substances that produce itching, hyperalgesia, and vasodilatation. Lewis named these nerves "nocifensor" and suggested that they belong to the posterior root system with efferent function. The flare around injuries and the burning vasodilatation are due to the stimulation of the fibers. As the reflex is not prevented by a nerve block central to the point of stimulation but is abolished by injecting the nerve distally to the point of stimulation, he believes that this is a peripheral effect within the distribution of a cutaneous nerve. This would be, then, a modification of the theory of an axon reflex, postulated previously by Lewis in the interpretation of the histamine flare (24).

Vasodilator impulses may reach the periphery, however, through the sympathetic nerves. J. H. Burn has reviewed the experimental evidence for sympathetic vasodilators. In man, Lewis and Pickering (25) have shown that reflex vasodilatation obtained through heating the trunk is abolished in the sympathectomized extremity.

It has been justly emphasized by Burn that the distribution of vasodilators varies in various species of animals. Clinicians have been frequently confused by "cat physiology." It is necessary, then, to examine the evidence we have from clinical observations relating to the mechanism of inflammatory or reactive hyperemia.

An anesthetic limb, such as results from a cut and degenerated peripheral nerve, from a spina bifida or syringomyelia, shows a negative histamine flare, a lack of inflammatory rubor and heat following infection. Such anesthetic limbs frequently show "trophic" ulcerations, which heal very slowly or not at all. The sympathetic pathway is intact but obviously does not contribute to a reflex vasodilatation. A sympathectomy for such "trophic" ulcers has never been of much benefit in our experience. An injury which, in addition to the soft parts, muscles, bones and tendons, produces a complete nerve injury, has never been reported to produce the syndrome of reflex dystrophy. In this group of patients suffering from sensory anesthesia, the afferent part of the reflex arc is interrupted and so are the parasympathetic efferent vasodilators as they run in the mixed nerves.

A paralyzed limb, such as a poliomyelitic extremity, may show a normal circulation but about 25 per cent of our cases (unpublished data) show cyanosis, mottling, chilblains, pressure sores. Sympathectomy in such cases warms the limb and abolishes the painful crises in cold weather. Reflex dystrophy has

never been seen in this group. The vascular changes are probably due to changes in the lateral horn, or demyelinization of the anterior roots, which may occur in poliomyelitis. The histamine flare and the reactive hyperemia are normal in such limbs.

The hemiplegic limb often shows an increased circulation early, owing to central sympathetic inhibition. Weiss and Ellis have studied such limbs in detail. In not a few cases there is a great deal of burning and throbbing pain in such limbs. It is customary to speak of a thalamic pain in such patients, with the admission that it is intractable. On the examination of such painful, burning, hemiplegic limbs, increased heat and osteoporosis may be observed. There seems to be a continuous vasodilatation present. If this is due to a mild chronic irritation of sympathetic centers or central pathways, a sympathetic block ought to give relief. Such an opportunity has not yet presented itself.

In patients with spinal arteriosclerosis, who concomitantly exhibit an obliterating arteriosclerosis in the extremities, there sometimes occurs a continuous burning pain with vasodilatation, a true erythromelalgia, which is aggravated by heat and improved by cold. All this may be present with pulseless arteries. Such nervous vasodilatation may be interpreted as an irritation of posterior roots or ganglia. A sympathetic block relieves the burning pain, a phenomenon which will be discussed in more detail later.

To summarize the reflex pathways which may be operating in reflex dystrophy, the afferent arc seems most likely to be in the somatic sensory nerves. It must be remembered that one of the earliest and most characteristic symptoms of Sudeck's atrophy is severe continuous or paroxysmal pain; the injury most frequently affects nerves, nerve sheaths, or periarticular structures, which are full of sensory receptors. The vasodilator efferent stimuli may go through the sympathetics but can be elicited in the absence of the sympathetic outflow, in which case they must be axon reflexes or spinal reflexes with an outflow through the posterior root vasodilators (Fig. 1). The excitation of efferent vasodilators produces substances at the end organs, which are responsible for both the vasodilation and the pain (Fig. 2). Ordinarily the choline-like substances, which are secreted at the end organs of parasympathetic fibers are rapidly destroyed; their persistence must either mean that the stimulation is continuous or that the esterase mechanism is defective (10). If this assumption is true, prostigmine or local acidosis definitely ought to aggravate the pain and vasodilatation of Sudeck's atrophy. This was true in Case 1 reported here.

The presence and gradual diffusion of this pain producing and vasodilator substance or substances confuse an orderly neurological examination. If the pain, hypoesthesia or

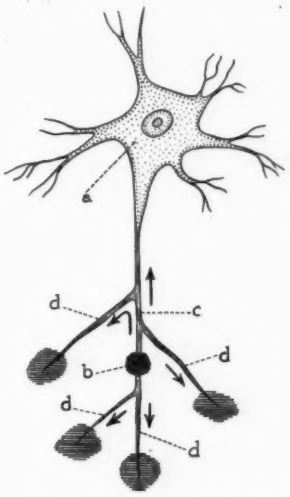


Fig. 2. The axon reflex illustrated in Figure 1 is emphasized in this diagram. Its persistence and gradual spread is responsible in our opinion for the syndrome of Sudeck's atrophy. *a*, Is the ganglion cell in the posterior root ganglion. *b*, Is the site of trauma. *c*, Represents an afferent sensory fiber whose stimulation results in pain and a vascular reflex is mediated through the cord as shown in Figure 1. *d*, Are the efferent vasodilator fibers whose stimulation results in a secretion of vasodilator substances. The shaded areas around the nerve endings indicate this diffusible, humoral substance. This is a diagram of F. Guillaume, in *Le sympathique et les systemes associés* published by Masson et Cie in 1921, which has been reinterpreted by us in the light of our present knowledge. In this diagram the stimulus is shown to spread centripetally to the next division whence it travels centrifugally; but it also travels in a centrifugal direction with the posterior efferent fibers. This type of spread would explain the observation of Tinel that blocking or sectioning of a peripheral nerve *distal* to the source of irritation may bring relief. This has been confirmed by Sir Thomas Lewis (23).

hyperesthesia of the affected limb is of a glove or stocking type, defying all patterns of segmental, somatic innervation, the neurologist will diagnose a functional disorder, a state of mind or a compensation neurosis. This has been our experience with every case of Sudeck's atrophy referred for a neurological examination.

We shall discuss later the peculiar autonomic imbalance from which some such patients seem to suffer but simply wish to state here that mainly because of the chemical mediation of the cholinergic parasympathetic efferent fibers, many neurologists simply ignore or evade this problem which not infrequently is presented before compensation boards.

THE DIAGNOSIS OF SUDECK'S ATROPHY

If the diagnosis of this syndrome had been made early enough many a long lasting disability would have been avoided. In studying the published reports in the literature and our own cases, we find that persistent pain of a burning character, with paroxysmal aggravations presented by patients whose injury is properly immobilized, noninfected, and seemingly on the way toward a normal course of repair, should make one suspicious of an early, beginning reflex dystrophy. In such a stage the extremity is warm, shows increased oscillations, edema of subcutaneous tissues and joint capsule; the muscles are hypertonic and are trying to splint the painful joint. There is no osteoporosis; this finding does not occur unless 4 to 8 weeks of continuous collateral hyperemia have been present. The pain is rather closely limited to the site of injury and its spreading character is not evident. We have already stated that periarticular structures are especially apt to give rise to such phenomena. Rauber (cited by Leriche) has counted 26 touch corpuscles around an interphalangeal joint. Nerves and blood vessels are equally apt to give rise to vascular reflexes when injured; but while Leriche and his followers have always emphasized vasoconstriction as the most frequent vascular phenomenon following injury, the burning pain of causalgia, the nutritional reflex of repair is a vasodilator phenomenon, which needs repeated emphasis and which will be demonstrated by our studies on blood flow.

The periarticular edema which may spread for quite a distance beyond the joint is the second characteristic phenomenon encountered early. Of course traumatic edema may be due to direct injury to veins and lymphatics, to pressure of casts and dressings, to infection or even to artificial constriction in cases of malingering. The edema of Sudeck's atrophy, however, is well localized around the site of injury and at first it is warm and flushed. Weeks later it may become hard, cyanotic and cold to touch. Because of the splinting action of muscles, and because of the synovial edema, the joints become stiff quite early and this is one of the most important residual disabilities with which one has to deal. Rheumatoid arthritis with vascular phenomena is quite analogous to Sudeck's atrophy.

There is finally an osteoporosis which, however, does not occur until several weeks after the injury. There is a spotty decalcification of the small bones of the hand or foot and in the metaphysis of long bones where vascularity of the bone is the greatest. We are able to show two biopsy specimens of such bones. There can be no doubt about the cause of this decalcification being due to hyperemia. Pommer, who has made a searching study of osteoporotic bone, quotes Herman Meyer who first stated in 1853 that a rapidly developing osteoporosis always means a hyperemia in the affected bone. Pommer made the interesting suggestion that Sudeck's atrophy may be due to hematomas in the marrow with collateral hyperemia but without actual bone injury. We had no opportunity to confirm or disprove this hypothesis.

The mottled, spotty bone atrophy later becomes diffuse, at which time it is indistinguishable from osteoporoses of other origin, such as the ones due to inactivity, senility, undernourishment, biliary fistula, and so forth. It must be emphasized that the diagnosis of Sudeck's atrophy should not be made on the basis of roentgen films. The syndrome may be present without it or it may be subsiding when the bone changes are at their height. Nor does the pain follow the course of

Fig. 3. Films of F. H., a 29 year old Mexican laborer, who suffered a fracture of the small bones of the left foot 15 months previously. Later a deep venous thrombosis developed. Causalgic symptoms appeared when weight bearing was resumed. Compensation neurosis was present. Gradual resumption of weight bearing, however, started recalcification. Note the coarse trabeculation in the heads of the left metatarsal bones. This case is typical of a Sudeck's osteoporosis complicated by atrophy due to disuse.

osteoporosis, because after sympathectomies the pain may rapidly subside but the osteoporosis persists for many months. The greatest value of roentgen films lies in serial examinations. When coarse trabeculation occurs with evidence of recalcification, the peak of disability has been passed. Complete restitution of structure has not been seen in our films.

DIFFERENTIAL DIAGNOSIS

The syndrome of Sudeck's atrophy may be so vague at first and clouded by additional complications later that its differentiation from other conditions is not always easy. In fact, the point should be made that it is often associated with the syndromes from which some authors try to separate it.

From the roentgenological standpoint and because of its frequency *atrophy of disuse* should be considered first. Atrophy of disuse is painless and not associated with marked vasomotor phenomena and certainly not with vasodilatation. Bone atrophy in the film is diffuse, comes on slowly and improves on the

Fig. 4. Case of A. McG. Left, normal control; right, tuberculosis of the ankle joint with marked bone atrophy. Note the destruction of the talocalcaneal joint.

assumption of active motion or weight bearing. It is our strong conviction that the early, spotty atrophy of Sudeck caused by increased blood flow to the bone and appearing within a month or so after the initial trauma is complicated later by a *superimposed* atrophy of disuse. At this stage a roentgenological differentiation is impossible. If the extremity can be made painless by procaine injections and adequate immobilization, the resumption of weight bearing and the active exercise of the surrounding musculature will hasten recalcification (Fig. 3).

Bone atrophy due to inflammation of joints, such as tuberculosis, may show increased blood flow, continuous pain, edema, and deformity. But the destruction of the talocalcaneal joint, seen on the right, leads to the diagnosis (Fig. 4). It may well be, however, that the bone atrophy seen around tuberculous and rheumatic joints is caused by the same vasodilator mechanism, which operates in the traumatic atrophy.

Aseptic necrosis of bone following vascular occlusions causes a rarefaction of bone, most visible in the vascular areas. Figure 5 shows such a film following the ligation of the brachial artery for a traumatic aneurysm. The necrotic areas do not have the same ground glass appearance as that seen in the acute traumatic atrophy of bone. The circulation, of

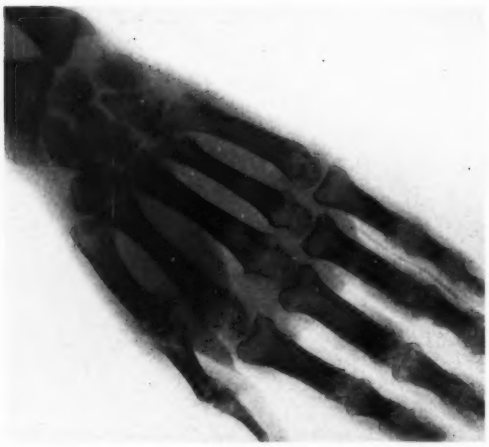


Fig. 5. Case of O. H. The brachial artery was ligated for a traumatic aneurysm 2 weeks prior to our examination and the taking of the films. Note the aseptic bone necrosis of the carpal bones and the metaphyses of the phalanges. The shafts of the phalanges are dense. The picture is entirely different from that of a Sudeck's atrophy.

course, is greatly diminished instead of being increased.

Senile osteoporosis involving primarily the trabeculae of spongy bone is most evident in the vertebrae; but it does involve other parts of the skeletal system. In 3 of our cases, Case 4, 5, and 9, there was a diffuse osteoporosis of the spine, far from the site of injury. It has been our impression that in such cases the local posttraumatic osteoporosis was more severe and, in addition, less responsive to treatment (Fig. 6).

Other forms of osteoporosis due to hyperparathyroidism, hyperthyroidism, pituitary basophilism, and adrenocortical syndromes, metastatic malignant lesions, multiple myeloma and osteomalacia have been discussed and differentiated from senile osteoporosis by Black, Ghormley and Camp. They can hardly be confused with Sudeck's atrophy.

The so called *traumatic flatfoot* which produces a great deal of pain on weight bearing, and because of lack of weight bearing may produce bone atrophy, must be differentiated from an ankle sprain with immediate, intractable pain, hypertonic and later atonic musculature with decalcification. Such a case is illustrated in Figure 7.

Traumatic neurosis, compensation neurosis, and malingering may have to be considered when patients are seen quite early. That the anxiety state of a patient may have a bearing on his autonomic reflexes is more than likely. In dogs, lesions in the motor area are usually followed by dilatation of vessels in the corresponding extremity, indicating that the dominant vasomotor representation in this part of the dog's cortex is vasoconstrictor (30).

Margaret Kennard removed the premotor area in monkeys and found that the mechanism for reflex vasodilatation was completely paralyzed. When such an animal was placed in a warm atmosphere the normal extremities showed quick vasodilatation but the affected extremity showed no such adjustment and remained much cooler than the control side.

In man the hemiplegic extremity shows interesting disturbances of heat regulation which have been studied by Bucy, Kerr and Underwood, Pilcher, Wyatt and Carney (29). The latter 3 authors demonstrated a unilateral overactivity of the sympathetic nervous system following a large infarction involving the distribution of the left middle cerebral artery. They interpreted this as a release phenomenon from cortical inhibition.

That autonomic reflexes are under cortical control is further exemplified by the work of Hesser, Langworthy and Kolb, who after decortication showed greater strength and consistency of gastric contractions. The stretch reflex showed delayed relaxation. They felt that reflex gastric activity is demonstrably controlled by cortical action.

From a standpoint of therapy all these disturbing cortical and subcortical influences should be eliminated as much as possible. Early settlement, reassurance, and relief from anxiety are all important before the local treatment is to be effective. Again it is our feeling that instead of trying to differentiate Sudeck's atrophy from a "state of mind," the participation of this element should be ascertained, as it probably plays a rôle in the symptoms of any injury.

A hard, traumatic edema of the hand is sometimes artificially produced by a constricting band. The constriction may not be visible to the naked eye at the time of examination but.

Fig. 6. Case of R. H., aged 71. Marked osteoporosis 15 months after injury of soft parts. This is Case 1 of our tabulated series. Severe osteoporosis of the spine was present.

if an infrared picture is available, it will show subcapillary extravasations in the form of a band (12).

THE COURSE OF THE DISEASE

There are mild types of reflex dystrophy which subside spontaneously in a few weeks or rapidly respond to treatment to be outlined. There are cases with moderate severity, causing complete disability, continuous pain, increasing contractures of tendons and joints and marked decalcification of bone. They run a self-limited course, however, and may heal spontaneously within a year. Often they leave residual stiffness, deformity and contractures of joints in spite of a very small injury. There is, finally, a severe form which shows a spreading neuralgia, a spreading osteoporosis, and gradually a mental fixation on this intractable lesion. Such patients may request amputation and threaten or commit suicide. A functional and economic rehabilitation of this group is hardly ever obtained. They may end up in sanitariums or closed institutions.

The severity of the original injury does not seem to determine the course of the disease. As a matter of fact, severe trauma causing fractures of long bones and total transection

Fig. 7. Diffuse osteoporosis, especially of the calcaneus and the tarsal bones with flattened longitudinal arch and spastic muscles, 7 months after a sprained ankle. The foot was never properly immobilized. It was warmer than its fellow and swollen. Nonpadded cast with walking caliper relieved the pain and permitted weight bearing. Recalcification was incomplete after another 6 months.

of nerves and blood vessels does not seem to be followed by this dystrophy. Sprains, mild frost bites, burns, partial nerve injuries, venous thromboses, low grade chronic infections, are in the history of these cases. One has the impression, after studying these patients, that their autonomic nervous system is labile, and hyperreactive. Whether this is the cause or the result of the prolonged disability we have not been able to determine. Most patients have been subjected to a neuropsychiatric study and this will be referred to in their histories. There has been, however, no single pattern or type of personality which was found to be characteristic for this syndrome. One cannot help referring to animal experiments which show that autonomic reflexes are readily elicited when central release is obtained (8). Applied to man this would mean that certain emotional states, anxiety, fear, and also uncontrolled pain are capable of exaggerating normally rapidly subsiding vascular reflexes. This problem is now being studied.

THE PREVENTION OF SUDECK'S ATROPHY

It is an interesting fact that men like Boehler, who strictly immobilize most injuries but encourage early weight bearing and active motion, have not seen or deemed worthy of mentioning the syndrome under discussion. In a small monograph, Karitzky has pointed out the importance of complete reduction of fractures, careful suture of torn muscles and tendons, and evacuation of hematomas to minimize the chronic stimulation from foci of

irritation. He especially emphasizes the surgical correction of torn muscles, tendons, and ligaments. In surveying our own material it has been obvious that minor injuries without injury to bone, but affecting ligaments, tendons, and musculature, are overwhelmingly present in the history of reflex dystrophies and large fractures, which are satisfactorily immobilized, are hardly ever followed by vasomotor and trophic disturbances.

The early and adequate control of pain, especially of pain which is hardly in keeping with the severity of the injury, is the next important preventive measure. Leriche has advocated the infiltration of hematomas in acute sprains wth procaine solution, a procedure which some men follow in this country. This is a simple and efficient method to prevent an activation of autonomic reflexes through painful sensory stimuli; if the block cannot be performed for any reason around the site of the injury, a nerve block proximal to the area such as that of the brachial plexus or the sciatic or femoral nerves is equally effective.

Whether early periarterial sympathectomy (16) or repeated sympathetic block with procaine (22) are more effective is difficult to say. The local infiltration is certainly more readily available to every surgeon dealing with minor injuries.

The third and last preventive measure is one which we mention with hesitation, although every experienced physician has made use of it for many centuries. Beside immobilization and relief from pain, the alleviation of fear, anxiety, the reassurance of the patient and the avoidance of all factors which may lead to a conscious or subconscious prolongation of a painful syndrome should be attempted. There is no clear cut evidence that the posttraumatic emotional status of a patient can actually influence the local autonomic reflexes originating from an injury; that it has an influence on the patient's reaction to anesthesia and to traumatic shock because of an activation of the autonomic nervous system is quite definite. Thus this syndrome may often be complicated or aggravated by a compensation neurosis, by a "state of mind," and this factor should be eliminated by the earliest possible settlement. A number of our patients (Cases 5, 13, 18, 23, 27, 28) have been regarded by competent neurologists as malingerers or hysterics; but it should be pointed out that the emotional status of the patient may actually influence his bodily reactions to injury. Thus the decorticated animal with an intact diencephalon, exhibiting sham rage shows many exaggerated autonomic reflexes. In man the typical histamine flare, an axon reflex, is markedly exaggerated under pentothal sodium anesthesia, whose depressant action is mainly central (unpublished data).

THE TREATMENT

As soon as an abnormal prolongation of pain, collateral hyperemia, muscle spasm, and fixation of joints is discovered, an attempt should be made to depress or interrupt the vascular reflex. If the site of irritation is clear, such as the presence of a partial nerve injury, a neuroma of an amputation stump, a scar in which nerves are embedded, or an arterial or venous thrombus, these foci may be excised. A torn muscle or ligament should be carefully repaired. This does not invariably break up the vicious cycle, especially if the syndrome is of several months or years duration. However, in Case 16 the resection of an obliterated segment of the axillary vein promptly relieved the edema and the vasomotor phenomena. In Case 14 the stripping of the iliofemoral vein was followed by complete relief of symptoms. In Case 23 the resection of a digital nerve which had been partially injured and acted as a trigger zone, gave relief from the spreading neuralgia but returned a few months later and progressed until other measures were taken. Generally speaking, our results with local excision of areas which were suspected of serving as chronic stimuli were good except in case of demonstrable nerve injuries. Thus the amputation neuromas, which are notoriously difficult to deal with, are apt to return with the pain spreading higher and higher. In patients who have already demonstrated their tendency to neuroma formation on section of the nerves, it is probably unwise to do any further peripheral nerve sections. On the upper extremity following poorly reduced or insufficiently im-

mobilized Colles' fracture one may see an involvement of the dorsal interosseous nerve with a typical trigger zone giving rise to severe reflex dystrophy (Case 24). The resection of this nerve has been suggested by Turner; however, it has been found unsatisfactory by Middleton.

Repeated local infiltrations of procaine and repeated injections to the paravertebral sympathetic chain as advocated by Leriche have given temporary relief and allowed the patient to tolerate physical therapy. It was found that if the patient received a block of the sympathetic chain, the joints would loosen up, the muscle spasm would relax and in this state heat, gentle massage, and active motion would bring about greater improvement. The repetition of this procedure two to three times a week has brought on great relief in Cases 20, 27, and 28.

In the later cases in the presence of much atrophy and contractures, a sympathetic ganglionectomy was done (Cases 4, 7, 8, 12, 17, 29, and 31). In certain patients (Cases 7, 12, and 17) this resulted in permanent relief from pain, although the contractures irreversibly remained. In others the neuralgia returned, spread higher and the pain became intractable. In such patients, posterior root section or chordotomy may fail as the source of the pain now has become thalamic. A surgical excision of the sensory areas in the cortex has not yet been done. Such patients often commit suicide and most of them become morphine addicts.

In summarizing our methods of treatment, they vary with the severity of the disease and with the stage in which they are seen. When the syndrome is recognized early, infiltrations of procaine are most helpful. If this fails or if the disease is of several months' duration, repeated procaine blocks of the paravertebral sympathetics have been done. Should this fail, a sympathetic ganglionectomy is indicated. Section of sensory nerves, dorsal roots or the spinothalamic tract has not been as useful as the interruption of the sympathetic fibers. Much attention is to be paid to the mental status of the patient. If there is a law suit pending, it should be settled at the earliest possible time.

STUDIES ON BLOOD FLOW

Several methods of determining blood flow in man have been described. While none of the methods is devoid of errors, we believe that the procedure of Abramson, which one of us (D.S.M.) has slightly modified, has eliminated many of the defects inherent in plethysmographs which operate by air instead of water displacement.

Our method of study was briefly as follows: the patient came to the laboratory early in the morning without breakfast. He was placed comfortably on a bed and rested for 30 minutes covered with a light woolen blanket. The temperature of the room, unless otherwise mentioned, was 28 degrees centigrade; the humidity was not controlled. The temperature of the water bath, in which the extremity was placed, was 32 degrees centigrade except in some studies in which cool and hot environmental effects were studied. A specially constructed plethysmograph was used in all observations (Fig. 8).

The principle of the machine, as that of all other plethysmographs, is to detect a volume change in the extremity by means of an occlusive pressure placed at the site of the insertion of the extremity into the machine. This occlusive pressure, for the upper extremity was 70 millimeters of mercury, and for the lower extremity 120 millimeters of mercury. The amount of occlusion, however, may be varied for different individuals, as has been discussed by Abramson. The change of volume of the extremity, produced by a sudden occlusion of venous outflow while the arterial inflow continues, is transmitted by a single system to a Brodie's bellows. This change in volume is recorded on a moving kymograph, by means of a small inker through which a small piece of hypodermic needle is inserted. A timer was used.

The apparatus can be used for the arm, hand, or foot. It has an entrance and an exit of equal dimensions, although one end can be closed securely by a special metal flange. The hand, foot, or forearm, after insertion into the machine, is surrounded by a rubber cuff which is glued in place by rubber cement. The hair is shaved off of the extremity, before insertion. The proper cuff is used with special care to prevent either constriction of the superficial vessels or to prevent the application of a very loose cuff which would allow leakage. This cuff is applied over the inner metal protrusion and secured in place by a monel metal ring with a large thumbscrew, which is used for tightening to prevent any water leakage. Horse saddle felt, one-half inch thick, is placed between the rubber dam and the outer metal plate after the extremity is inserted. This immobilizes the hand in place rather securely. The hand or foot rests in the double cylinder comfortably on a copper sling so that the extremity is not in contact with the sides of the inner walls. For the hand or forearm the double cylinder is used, whereas the

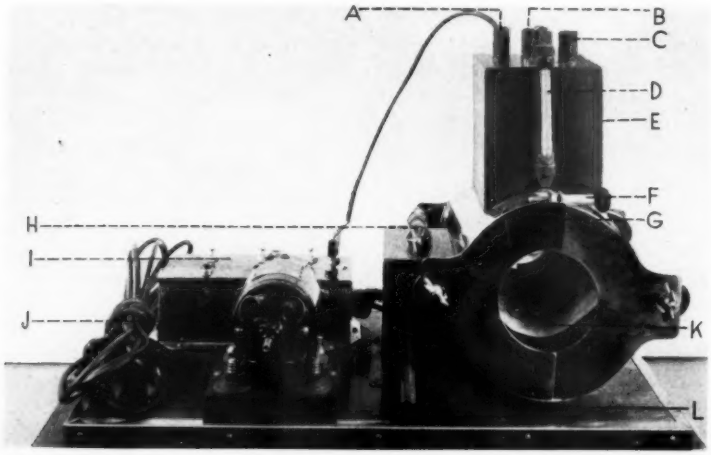


Fig. 8. Final design of plethysmograph; the result of several modifications. *A*, insertion for electroregulator; *B*, outlet leading to kymograph; *C*, outlet for thermometer; *D*, indicator to show the amount of water present in the plethysmograph; *E*, cubicle for foot; *F*, thumbscrew attached to monel metal ring to tighten rubber dam; *G*, metal panel encircling hand or foot; *H*, plunger to calibrate blood flow after each determination; *I*, switchbox; *J*, motor; *K*, suspension for extremity; *L*, outlet.

cylindrical top of the plethysmograph has been applied to the foot and may take a foot up to size twelve with relative comfort. An electric stirrer with four blades attached through the wall of the machine by a coupling rod to a motor with reduced gear and an electric heating element directly attached to a thermostat in the machine are placed in a small metal cubicle off to one side of the main plethysmograph. They are in direct contact with the water that surrounds the limb. The fan is routinely turned on for all experiments, whereas, the heating element is used when an elevated water medium is necessary. The fan distributes the water after the heating element is turned on so that one may be sure of a constant, uniform change in temperature at all times. There are three openings on top of the machine.

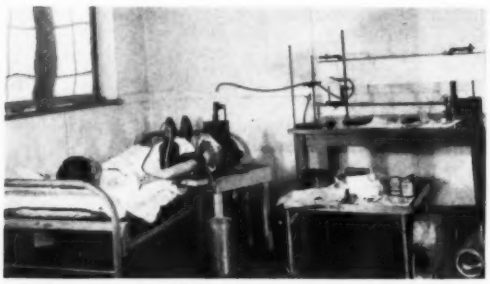


Fig. 9. Blood flow determination on the right hand. The kymograph with ink recorder is to the right.

One is used for a thermoregulator. The second opening, the largest one, which is 1 inch in diameter and in the center, directly connects the machine to the volume recorder, while the third opening is used for the insertion of a small thermometer. One may read the temperature of the water at any moment during the experiments. Water at 32 degrees centigrade is added into the machine through one of the top openings. The machine must be entirely filled and tapped gently to release any hidden air bubbles that may occur in one of the four corners of the upper cylinder of the apparatus. When the extremity is surrounded with the cuff and secured by cement attached to a rubber dam ($\frac{1}{32}$ inch thick rubber), it is then secured by the monel metal ring, tightened by a thumbscrew to prevent water leaks and again reinforced by the felt and the special metal plates. The machine now, after filling it with water is ready to be used for determinations of blood flow. A small blood pressure cuff of 5 centimeter width is placed about the extremity, close to the entrance of the machine. The cuff is directly attached to a mercury manometer which has been previously calibrated and which indicates the amount of occlusion applied. The motor is turned on, rotating the fan, and the thermoregulator adjusted as necessary to change the temperature of the water. From 5 to 7 minutes are usually required for every 5 degree increase in temperature of the water. The subject for the experiment is well covered, exposing only one extremity at a time, the other being kept covered during the time of the experiment. Absolute rest is imperative and no external stimuli should interfere with the patient

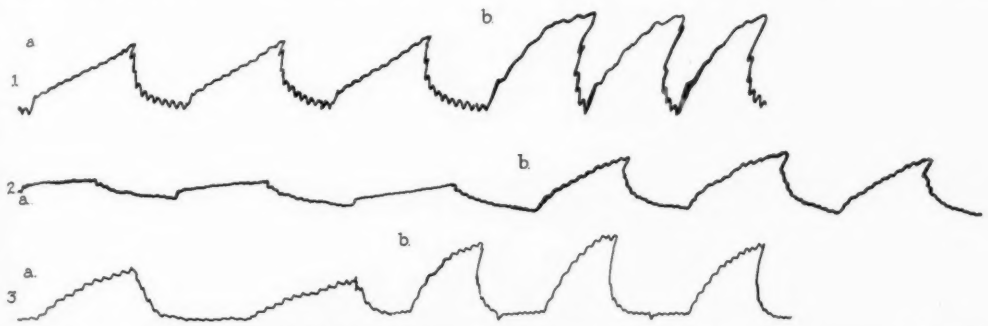


Fig. 10. *1a*, G. D. (Case 12). Blood flow left normal hand. Nine trials averaged 3.4 cubic centimeters blood per 100 cubic centimeters limb volume. *1b*, Blood flow involved hand. Ten trials averaged 5.9 cubic centimeters blood per 100 cubic centimeters limb volume. *2a*, R. D., Case 8. Blood flow left normal foot. Nine trials averaged 2.13 cubic centimeters blood per 100 cubic centimeters limb volume. *2b*, Blood flow involved foot. Six trials averaged 5.6 cubic centimeters blood per 100 cubic centimeters limb volume. *3a*, B. S., Case 6. Blood flow right normal hand. Ten trials averaged 2.61 cubic centimeters blood per 100 cubic centimeters limb volume. *3b*, Blood flow involved left hand. Fifteen trials averaged 2.9 cubic centimeters per 100 cubic centimeters limb volume.

during the course of the experiment (Fig. 9). A 30 minute rest period is allowed before each test and 60 second intervals are allowed between each succeeding occlusive pressure. The position of the extremities is most important. For the lower extremity the patient lies comfortably in bed, the hip abducted 10 to 15 degrees and the leg and foot elevated about 4 inches above heart level. All muscles are in a relaxed position. For the upper extremity the shoulder is abducted to 70 degrees, the elbow flexed to 140 degrees and the hand kept either in pronation or in midsupination-pronation, as this is the most comfortable position for the patient to assume for prolonged periods. Special blocks with grooved out areas for the reception of the calf muscles, as well as blocks for the humerus have been made to elevate the lower extremity comfortably before entrance into the machine. A small pillow is used for the head and neck.

Each determination included a blood flow reading on the normal, opposite extremity. The cuff was placed at exactly the same position and exactly the same location as the normal extremity. It was important to determine whether there was any occluding pressure before the test was made by detecting any change in color of the extremity or by

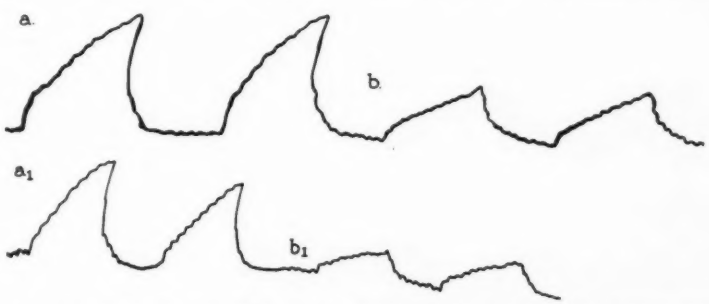


Fig. 11. R. H., Case 1. This chart indicates the blood flow in the normal and involved hands with respective blood flow responses to a decreased temperature of the water medium (15 to 17 degrees C.). *a*, Blood flow of involved right hand averaged, in 7 trials, 8.7 cubic centimeters of blood per 100 cubic centimeters of limb volume. *b*, Exposure to decreased temperature of 15 degrees—17 degrees averaged in 6 trials 5.3 cubic centimeters of blood per 100 cubic centimeters of limb volume. a_1, blood flow of normal left hand in 5 trials averaged 8.6 cubic centimeters of blood per 100 cubic centimeters of limb volume. b_1, Blood flow of normal hand after exposure to 15 to 17 degrees C. water medium in 5 trials average 2.7 cubic centimeters of blood per 100 cubic centimeters of limb volume. Note that the response of the injured extremity to cold is sluggish compared to the response of the normal extremity.

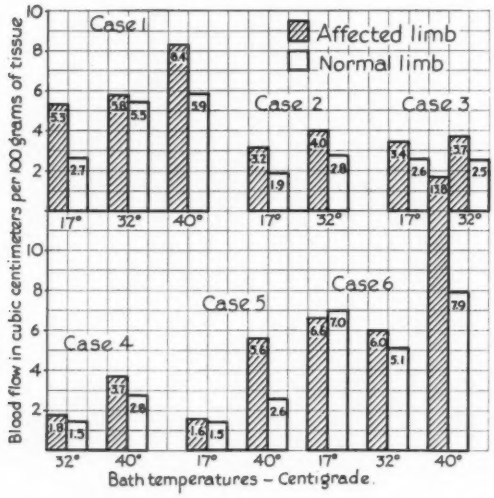


Fig. 12. The effect of temperature on blood flow in Sudeck's atrophy.

TABLE I.—STUDIES OF BLOOD FLOW IN POST-TRAUMATIC REFLEX DYSTROPHY

Case	No. of studies	Blood flow in c.c. per 100 c.c. of limb volume		Limb volume in c.c.		Percentual Increase of blood flow in affected limb
		Affected limb	Normal limb	Affected limb	Normal limb	
1 R.H.	13	5.85	4.84	444	432	+20
2 S.O.	10	4.0	2.84	1013	934	+30
3 F.O.	6	3.41	2.55	975	930	+33
4 K.W.	6	1.9	1.43	860	896	+35
5 M.B.	12	1.58	1.50	838	768	+ 5
6 B.S.	13	6.07	5.11	240	270	+18
7 C.B.	4	1.0	1.23	887	896	−23
8 R.D.	8	3.7	1.8	916	849	+48.6
9 E.L.	9	7.4	5.92	258	282	+25
10 B.M.	10	5.86	4.60	463	463	+29
11 M.Y.	11	4.49	2.80	878	826	+60
12 G.D.	12	5.12	4.0	935	881	+28
Total	114	45.26	38.42	8707	8427	+30.3

the complaints of the patient. The machine was allowed to run for several minutes to determine a standard base line. Artefacts were watched for and were detected during the course of the experiment and results obtained therefrom were tested or corrected. Sudden rises in the curve, too flat a plateau or various dips or sudden elevations in the curve meant either a defect in the machine, a leak in the system, or a movement on the part of the patient. The patients were told not to cough, sneeze, or move. They were also told not to fall asleep or move their other extremities in any way. Leaks were the most annoying problem but could be controlled with an exact application of the rubber to the wrist or ankle and a proper closure of the constricting ring over the inner tube of the machine. The usual curve was that of a slow rise and a relatively flat plateau after some time elapsed following occlusion of the involved extremity (Figs. 10 and 11). Pulse waves, as seen on the kymograph, were an absolute necessity to determine the status of the machine. Calculations were made on the determined curves by injecting 2 cubic centimeters of water directly into the system through a needle and rubber tubing at the exit of the machine. Another injection of the same quantity of water, as well as a third, if this was necessary, was made to calibrate the inflow of blood as determined by the rise of the curve. These injections were made because it was found that the apparatus was sensitive to a different degree at different levels of the curve.

The results were calculated in cubic centimeter of blood per 100 cubic centimeters of limb volume. From 6 to 20 trials were made on each extremity and the average taken from them. The room temperature, bath temperature, and blood pressure were noted at each sitting.

Aside from the plethysmographic studies, oscillometric readings were done in all subjects. Pachon's oscillometer was used. Temperatures of the digits and of the toes were made for comparative studies in some cases before and after sympathectomy. A General Electric apparatus was used for these determinations. Blood chemistry studies, calcium, phosphorus, and phosphatase, total proteins and uric acids were determined. Also urine and blood counts were done at the same time. Injections of the stellate ganglion and lumbar ganglionated chains were made with novocain, usually before operation to determine the effect of blocking the vasomotor response to the extremity.

RESULTS OF BLOOD FLOW STUDIES

A total of 114 determinations were made on the affected and the same number on the opposite extremity of 12 patients. Table I shows a brief summary of our findings. It is noteworthy that in 11 of 12 patients, there was increased blood flow in the affected limb, varying from 60 per cent to as little as 5 per cent. The volume of the affected limb was also usually larger, although this increase did not, by any means, parallel the marked increases in blood flow. The only patient who showed a decreased flow in the affected limb was C. B., Case 7, who had a very small rest-

ing blood flow and who, as a subsequent sympathectomy showed, must have had marked peripheral vasoconstriction. While his basal metabolic rate is not available, his clinical findings were those of a hypothyroidism which is usually characterized by decreased blood flow through the extremities.

In Case 5, in which there was no definite increase in blood flow, the following status of the affected extremity was found: the first determinations of blood flow were made 10 months after a fracture of the fibula, which was immobilized in an equinus position. The affected extremity showed marked trophic changes. The skin was shiny and atrophic, the nails were ribbed, and the ankle joint was swollen. There was also some increase of hair on this extremity. In the 12 blood flow determinations, the average of which is given in Table I, 6 gave a higher and 6 gave a lower reading than that of the control extremity. All readings were quite low. This patient no doubt represents a late stage of Sudeck's atrophy with marked organic changes.

C. B., Case 7, had an automobile injury to the left ankle, following which no fracture could be demonstrated. The basal blood flow of this patient was exceptionally low, although he was not arteriosclerotic and only 37 years old. He was always cold, wore heavy underwear always, and responded poorly to decreases in environmental temperature. Unfortunately no basal metabolism test was obtained. In 9 of the 12 determinations the blood flow was higher on the affected extremity, but most of these were done after a lumbar sympathectomy. The average reading of the 4 determinations done before sympathectomy was only 1 cubic centimeter per 100 cubic centimeters of limb volume; this being the lowest value obtained in this series and closely approximating the blood flows obtained by us in advanced obliterating arteriosclerosis.

With the exception of these 2 cases, there was a definite increase in blood flow of the injured extremity. The average increase of the 12 cases was slightly over 30 per cent. This remarkable increase, persisting over many months or several years after the injury, is the most characteristic feature of the syndrome.

TABLE II.—THE EFFECT OF SYMPATHECTOMY ON BLOOD FLOW IN REFLEX DYSTROPHY

Case		Blood flow in c.c. per 100 c.c. of limb volume		Number of determinations
		Affected limb	Normal limb	
4 K.W.	Before operation	1.9	1.43	6
	After operation	3.7	3.2	3
7 C.B.	Before operation	1	1.1	4
	After operation	2.68	1.9	8
8 R.D.	Before operation	3.7	1.87	8
	After operation	3.52	2.4	5
12 G.D.	Before operation	5.1	4.06	5
	After operation	4.4	3.85	2
	Totals Before	11.7	8.46	23
	Totals After	14.3	11.35	18

Its possible mechanism has been discussed under the heading of vasodilator reflexes. Of great interest was the behavior of such extremities when they were subjected to changes in environmental temperature, while the room temperature was kept at 28 degrees centigrade and the temperature of the water bath was varied from 15 degrees centigrade to 40 degrees centigrade. Figure 12 shows that at lower temperatures the difference in blood flow between the two sides was generally not as marked as at higher temperatures, but, in addition, the affected extremity did not respond with vasoconstriction as readily in a cooler water bath as did the normal limb. One would expect such findings in the presence of a chronic vasodilation; but, as pointed out, there is often a masked vasoconstriction in such limbs, which may account for the instability of response seen in Case 1.

THE EFFECT OF SYMPATHECTOMY ON BLOOD FLOW

Of several patients who underwent sympathectomy, we have blood flow determinations, before and after operation, on 4 (Table II). K. W., Case 4, was seen 5 months after a fracture of the fibula and a severe sprain of the ankle. Severe, continuous pain occurred immediately. A cast was applied for 2 months. Because of the marked osteoporosis (Fig. 13) and the early intractable pain, a diagnosis of Sudeck's atrophy was made by one of us

Fig. 13. Severe diffuse osteoporosis in K. W., Case 4, 5 months after a sprained ankle with a fracture of the fibula.

(D.S.M.), when seen 5 months after the injury. The bimalleolar space was widened, and the foot was held in an equinus position. The ankle was cold, swollen, and the skin was shiny. In 6 determinations of blood flow, before sympathectomy, there was an increase in 4 out of 6. There was a small, plateau-type of blood flow curve as seen in obliterative vascular diseases. The Wassermann reaction was strongly positive in spite of intermittent treatment with neoarsphenamine and bismuth. Following sympathectomy, there was an increase in blood flow in both extremities, a finding which Leriche has indicated for many years. While the pain has disappeared and the patient has been able to bear weight on the extremity, the osteoporosis is still present and is even detectable in the knee and hip of the same side.

Case 7, C. B., has already been referred to as having a very low preoperative blood flow. The left, injured ankle was much warmer than its fellow, although it felt colder to the patient. Four readings were done before and 8 readings after a left lumbar sympathectomy. The bath temperatures were kept constant at 32 degrees centigrade. The postoperative readings were made from 11 days to 3 months after operation. The readings were consistently higher after sympathectomy. Clinically, the limb operated upon was dry, warm, and pain on weight bearing disappeared.

Case 8, R. D., dislocated her left foot with a marked extravasation of blood at the external malleolus. Following reduction, the foot and lower leg were placed in a cast for 6 weeks. Eight blood flows were done from 7 months to 11 months after the accident, indicating a marked increase on the injured side. Six blood flows were determined from 11 days to 4 months after sympathectomy. While there was complete relief from pain, the blood flows were not especially affected on the injured side where vasodilation was already marked.

Case 12, G.D., had been seen comparatively early, 10 weeks after an injury to the left ankle without fracture. While she was properly splinted, continuous intractable pain developed immediately after the injury. The blood flows were high in both feet but definitely higher on the injured side. After sympathectomy the blood flows decreased on the side operated upon and not on the control side. Clinically there was marked relief from pain. A shortening of the Achilles tendon had to be corrected by casts.

In summary, a total of 23 blood flows were done prior and 18 blood flows following sympathectomy. The interpretation of the paradoxical findings of the changes in blood flow in relation to the clinical improvement will be attempted in the discussion.

STUDIES ON CALCIUM METABOLISM

Blood calcium, phosphorus and phosphatase determinations were made on 12 patients affected with reflex dystrophy (Fig. 14). In 4 patients the determinations were made twice several weeks apart. While the calcium and phosphorus showed no deviation from the normal, the phosphatase was slightly but definitely elevated in most instances. The blood phosphatase level for the normal adult should not exceed 4 Bodansky units. The lowest figure of 4 units was obtained in Case 10, B.H., who suffered a blunt injury to the dorsum of the hand and developed a spotty atrophy of the carpal bones, not a great mass of bony structure. The highest reading of 10.4 units was obtained in Case 9. This patient had a diffuse osteoporosis of the entire right upper extremity, which followed a fracture of the shoulder 6 years previously. This woman was 58 years old and her traumatic osteo-

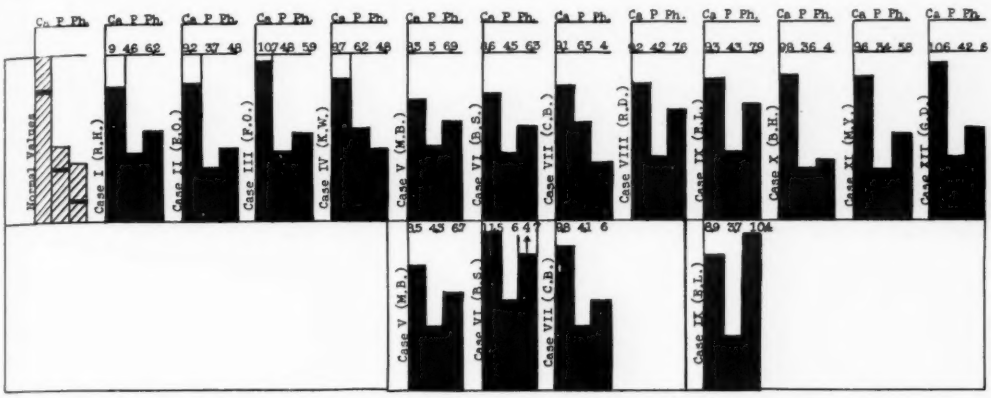


Fig. 14. Calcium, phosphorus and phosphatase studies in Sudeck's atrophy. Normal values: calcium, 9.5—11 mgm. per 100 c.c. serum; phosphorus, 3.7—5 mgm. per 100 c.c. serum; phosphatase, 1.5— 4 units (adult).

porosis may well have merged with a senile osteoporosis, a complication which will be discussed in our comments.

There are not many control determinations on other forms of atrophy but from our figures, it would seem that there is some correlation between the extent of bone atrophy and height of the blood phosphatase level.

BONE BIOPSIES

In K.W., Case 4, during a lumbar sympathectomy a biopsy specimen was taken of the lower end of the tibia. The original injury was a dislocated ankle with a fracture of the lower end of the fibula 5 months previously. The soft tissues were vascular when the bone was exposed. The bone was purplish-red in color and the cortex was very thin. This finding reminded us of the suggestion of Pommer, one of the foremost students of osteoporosis, that Sudeck's atrophy might be initiated by intramedullary hemorrhages. The medullary canal was unusually wide; on chiseling through the cortex a marked hemorrhage ensued which was finally controlled by pressure. The piece of cortical bone removed was extremely thin and fragile. The microscopic sections revealed a bone atrophy with reduced spicula. There was marked osteoblastic activity around each bone island and a marked increase in vascularity. The periosteum was intact and markedly thickened (Figs. 15 and 16).

In E.C., Case 9, suffering from a reflex dystrophy of 6 years' duration, a biopsy specimen was removed from the lower end of the ulna. The initial trauma was a fracture around the shoulder joint. The cortex was thin and the medullary canal was wide, but only a moderate amount of bleeding was encountered. The histological section revealed a simple atrophy, no increase in blood supply and reduction of osteoblasts. Compared with the previous case, this was a late stage of Sudeck's atrophy (Fig. 17), an atrophy of disuse.

DISCUSSION OF CASES

We have detailed records on 33 patients whose essential data have been summarized in Table III. As discussed earlier in this paper, the diagnosis of Sudeck's atrophy was not made unless the following criteria were present: (1) history of injury (or in some cases, vascular thrombosis); (2) almost instant, continuous severe pain, which does not subside in a few days and is out of proportion to the detectable damage to tissues; and (3) a spotty and later diffuse osteoporosis, which, however, is not present in the first 4 to 6 weeks after the initial trauma.

The table reveals that no age is exempt. In a few older individuals, the differential diagnosis between a traumatic and a senile osteoporosis becomes difficult. In Cases 4, 5, and 9, x-ray films of the spine revealed a

TABLE III.—REFLEX DYSTROPHY OF THE EXTREMITIES

Essential Features in 33 Cases

Case	Age	Initial trauma	Duration of syndrome in months	Treatment	Result
1 R.M.	71	Injured shoulder	9	Spica cast	Gradual improvement, ankylosis
2 E. O.	54	Sprained ankle	8	Molded splint	Gradual improvement, ankylosis
3 F.O.	60	Sprained ankle	2	Unna's paste by chiropodist	Painful ankylosis
4 K.W.	49	Dislocated ankle, fracture of fibula	5	Cast for 2 mos., lumbar sympathectomy	Marked improvement, sympathectomy incomplete
5 M.B.	46	Sprained ankle and fractured fibula	24	Refused	Total disability
6 B.S.	43	Fracture of navicular bone	18	Cast, manipulation of elbow, shoulder	Gradual improvement
7 C.B.	37	Sprained ankle	4	Lumbar sympathectomy	Excellent
8 R.D.	27	Dislocated ankle	7	Reduction, cast, sympathectomy	Excellent
9 E.L.	58	Fractured shoulder	78	Immobilization, periarterial sympathectomy	Improved
10 B.H.	49	Sprained wrist	1	Strapping, stellate block	Excellent
11 M.Y.	58	Sprained ankle	4	Cast	Unknown
12 G.D.	39	Sprained ankle	3	Splint, lumbar sympathectomy	Excellent
13 J.G.	18	Wringer injury at wrist	7	Periarterial sympathectomy	Excellent
14 J.N.	41	Iliac periphlebitis	36	Stripping of vein	Excellent
15 D.R.	25	Nodular phlebitis	36	Refused	Unknown
16 H.S.	25	Thrombosis on effort	8	Stripping of vein	Excellent
17 B.H.	38	Infection and incision over inner ankle	5	Lumbar sympathectomy	Relief from pain, ankylosis
18 E.H.	29	Wringer injury at wrist	36	None	In State institution
19 R.C.	?	Sprained ankle	12	Cast, strapping	Poor
20 E.D.	28	Sprained wrist	3	Stellate block, physiotherapy	Excellent
21 J.Y.	50	Crushed 5th finger	12	Alcohol block to stellate	Excellent for 4 mos., recurrence
22 S.C.	48	Crushed 2d finger	6	Repeated procaine block	Fair
23 E.H.	28	Fractured phalanx	6	Periarterial sympathectomy	Slow recovery
24 J.C.	40	Colles' fracture, poor reduction	12	Repeated local and stellate block with procaine	Fair, ankylosis
25 H.M.	30	Hematoma of dorsum of foot	5	Lumbar sympathectomy	Excellent for 1 year, recurrence
26 F.H.	37	Deep thrombophlebitis, fracture of tarsal bones	20	None	Unknown
27 D.H.	20	Blunt injury to dorsum of hand	1	Repeated local and stellate block	Excellent
28 L.S.	34	Blunt soft tissue injury to dorsum of hand	1	Repeated stellate block	Excellent
29 J.Y.	47	Blunt injury to tibia, no fracture	14	Venous hyperemia, lumbar sympathectomy, theelin	Excellent
30 J.D.	64	Sprained ankle	42	Physiotherapy, orthopedic support, incomplete sympathectomy	Fair
31 A.C.	60	Crushing injury to three fingers	48	Anterior preganglionic sympathectomy	Excellent
32 H.J.	64	Sprained ankle	8	Walking cast	Excellent
33 R.A.	19	Scar following incision of toe	6	None	Gradually subsided

*Cases 13 to 17 have been published previously (*Arch. Surg.*, 1937, 34: 939).

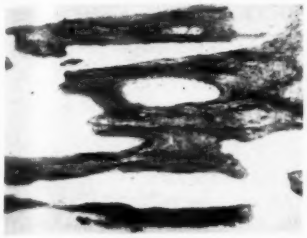


Fig. 15.

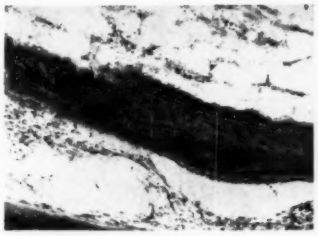


Fig. 16.

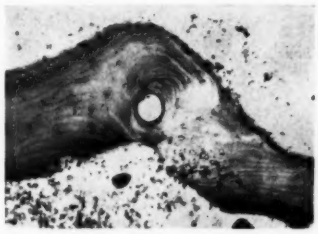


Fig. 17.

Fig. 15. Photomicrograph of lower end of tibia in the case of K. W. The general structure is that of bone atrophy although the adult bone cells may be seen in the tissue. There is evidence of degenerative nuclei in some bone cell spaces.

Fig. 16. Photomicrograph of case of K. W. near the marrow cavity. Bone cells may be seen adjacent to the marrow. This section indicates a more active section than that of Figure 15.

Fig. 17. Case of E. L. This is a bone section taken from the upper third of the ulna in a 7 year old case of Sudeck's bone atrophy. The bone is atrophic in nature, adult bone cells being seen in lacunar spaces with the nuclei at the periphery and with evidence of degeneration.

diffuse senile osteoporosis. Yet their local injury and the character of the spreading neuralgia from a comparatively small painful focus made the diagnosis of a traumatic dystrophy necessary.

In a case not tabulated because of meager data, a 12 year old girl, who suffered a fracture of the ankle with malleolar separation, developed an acute and quite extensive decalcification with severe pain both with and without weight bearing. This occurred in spite of the fact that she had been allowed early weight bearing with a walking caliper. Obviously the immobilization was incomplete.

In Cases 10, 20, 27, and 28, the syndrome was only a few weeks' duration. However, they were not mild cases. Cases 27 and 28 were under the close attention of one of us (G. de T.) and required a great deal of sedation before the procaine injection started. The local infiltration of procaine at the site of maximal pain seemed to alleviate the pain for a few hours. The block of the stellate ganglion gave more lasting relief, lasting for a week or more; the second or third injection usually stopped the process.

Cases 9, 13, and 23 had a periarterial stripping. Of these 13 and 23 were comparatively early cases. They both recovered but their recovery was slow and it is now our impression that these patients would have done better with repeated paravertebral injections of procaine. In Case 9, a difficult case of 6 years' duration, with a great deal of trophic changes in the skin, tendons, and joints, patient had only a partial benefit from this operation, which was done elsewhere.

Cases 14 and 16 had a perivenous stripping for venous thromboses which resulted in reflex vasomotor and neuralgic phenomena. Both patients obtained lasting (4 and 5 years') relief.

Cases 4, 7, 8, 12, and 17 had lumbar sympathectomies. In Case 4, the lumbar sympathectomy, done elsewhere, resulted in incomplete denervation; the area of greatest sensitivity showed extensive sweating. Nevertheless, she showed slow improvement. The 4 other cases had early relief from pain, in spite of the fact that vasodilation and osteoporosis remained for some time. These were complete sympathectomies. As all 4 were severe and late cases, the orthopedic management of joints and tendons needed special care. Two of the patients' ankles were in equinovarus position and were corrected by lengthening of the Achilles tendon and casts. All such secondary complications could, of course, be readily avoided by early management.

With the exception of a few injured shoulders, Cases 1, 9, and 29, in which injury to ligaments, tendons, and brachial plexus is so frequent, most of the injuries occurred at the hand, wrist, or foot and ankle; fractures were rare and mostly associated with ligamentous tears. In some instances direct irritation to nerve trunks was demonstrable. Thus, in Case 13, that of J. G., whose wrist was

caught in a wringer, exploration of the wrist revealed a perineural fibrosis of the median nerve. In Case 8, R.D., when she was examined 7 months after a severe sprain of the ankle, there was a plantar anesthesia with a loss of position sense of the third, fourth, and fifth toes. In the case of B. H., a farmer, who drained an abscess over the inner malleolus, a small strip of hyperesthesia was present corresponding to the area of the saphenous nerve. In Case 24, J. M. C., a poorly reduced Colles' fracture must have injured the dorsal interosseous nerve, as radiation of pain along its area of distribution could be elicited. In Cases 27 and 28 both patients suffered a mild injury to the dorsum of the hand and partial injury to the superficial radial branch was well established. In a case seen recently in consultation the area of the partially traumatized anterior tibial nerve compressed by a callus could be mapped out by a skinarea of increased temperature. As these injuries were mostly partial and consisted of perineural hematomas or stretching, their diagnosis could only be made when seen early after injury. The significance of these minor partial injuries to sensory nerves will be discussed under "Analytical Comments."

ILLUSTRATIVE CASE REPORTS

Early severe cases difficult to differentiate from psychoneurosis or hysteria

D. H., Case 26, was a student nurse who sustained a mild blunt injury to the dorsum of the left hand. The hand had been caught between a stretcher and a cement wall. Immediately severe pain developed. There was no fracture, no evidence of soft tissue injury, and no hematoma. Immobilization on a splint for 1 week gave no relief. Physical therapy, consisting of heat, whirlpool, and massage aggravated the pain. When seen 2 weeks after the injury, the hand was slightly swollen. The wrist and fingers were held in rigid extension. The extensor muscles were spastic and permitted no movement. A few cubic centimeters of procaine injected in the region of the dorsal interosseous nerve promptly relaxed the spasm; the patient could close her fist readily. The effect would only last a few hours after which the same rigid position was assumed. Daily injections of procaine in and around the same area gradually produced improvement of the hand but a spreading pain developed in the region of the elbow and shoulder. These were controlled by two injections into the stellate ganglion. After 3 weeks of treatment, the hand was painless, completely regained function and showed no swelling.

The patient's parents were dead; she had a guardian with whom she had many conflicts. Her mother died in an insane asylum. Neuropsychiatric examination revealed a very unstable individual and a "hysterical hand" was suggested. The patient had many other functional complaints: severe pylorospasm, irritable bowel, and inability to swallow at times.

The differential diagnosis between a true hysteria and an early reflex dystrophy seems impossible in this case. If the procaine injections acted as mere psychotherapy, the approach seemed easy and the treatment short. A close check on her later behavior showed that there has been no recurrence of this syndrome for 6 months.

Another early severe case is that of L. S., Case 27.

This patient's hand was injured in a streetcar accident. When seen a month later, she was first urged to obtain a settlement as a compensation neurosis had to be ruled out. She was a highly excitable woman with a low pain threshold, as demonstrated by the Libman test. There was no swelling, no tendon or nerve injury, and no fracture. There was, however, an exceedingly tender area just proximal to the wrist on the dorsoradial side. When pressure was exerted over this area radiation of pain was felt over the area of the superficial radial nerve, both proximally and distally. There was much more spreading neuralgia in this instance than in the preceding one. The shoulder, the back of the neck and the anterior chest wall were sensitive. Two injections of procaine into the second and third dorsal sympathetic ganglia relieved the pain. She was reexamined a month later and had no recurrence of symptoms.

Both of these cases were seen too early to show osteoporosis and neither was there any trophic change in the soft tissues. The oscillometric curves and histamine flares, however, indicated an increased blood flow to the affected area; a partial injury or a perineural hematoma of the dorsal interosseous nerve was a distinct possibility. This is the explanation given by Turner many years ago for trophic lesions following Colles' fracture. This mechanism reminds one of the "hysterical ankle" of some patients, who after a minor sprain complain of intense burning pain with paroxysmal aggravation, and a proximally spreading neuralgia. We have seen such a patient but no study was made at that time of the patient's circulation.

Early mild case

B. H., a 49 year old white male struck a man on the head with the palm of the left hand. Within a few hours marked swelling of the dorsum of the hand and wrist developed. X-ray films were negative for fracture. He received an adhesive tape dressing for 2 weeks followed by heat and massage at home. There was a continuous pain and burning which was relieved by cold and exaggerated by heat. Five blood flows out of 6, the first one being determined a month after the injury, showed a definite increase on the injured side (Case 10, Table I). Two oscillometric recordings were made at a 3 day interval. The first indicated a marked increase in oscillations, the second showed no difference between the 2 sides at the wrist and lower arm. Histamine flares were equal. X-ray films indicated an early spotty atrophy of the carpal bones. Within another month the pain and edema disappeared and the function of the arm returned three months following the injury.

In this patient no spreading neuralgia developed and regression of symptoms started within the first few weeks after injury.

Intermediate case in state of regression complicated by compensation neurosis

F. H., a Mexican railroad laborer, suffered a fracture of the tarsal bones of the left foot. A cast was applied but a venous thrombosis of the deep veins of the lower leg necessitated the removal of the cast. He had severe burning pain in the leg and foot from the first few days on. He was immobilized off and on and received a great deal of physical therapy. His pain continued and the leg swelled each time he started to work. His lawyer started suit against the railroad. On examination, 1½ years after the injury, the left leg was moderately edematous and weight bearing was painful. The patient spoke poor English but his anxiety to obtain the maximum benefit out of his disability was obvious. X-ray films showed a mild osteoporosis in a stage of recalcification with coarse trabeculation (Fig. 3). The peak of the osteoporosis had obviously been passed. It was felt that by obtaining a settlement and with the use of a nonpadded plaster cast his symptoms would be ameliorated. Six months after being seen he was working on another job, well satisfied, with a moderate painless edema.

Early severe case treated by sympathectomy

G. D., a 39 year old colored woman, fell down two steps and sprained her ankle. X-ray films indicated no fracture. She was placed in a splint for 3 weeks during which time she had severe pain which would awaken her at night or come on during complete rest. Repeated X-ray examination revealed osteoporosis and again no fracture was found. (Fig. 18). In spite of heat, massage, and soaks, she continued to have pain for 2 months, after which she was admitted for study. The left ankle was markedly swollen; pain was diffuse, but most of the tenderness occurred on pressure below the external malleolus in the region of the lateral plantar nerve. This ankle was from 2 to 2.5 centigrades warmer than the opposite ankle. The oscillometric curve was higher on this side than the other. Four blood flow determinations indicated a marked increase of the affected side, a 28 per cent increase on the average (Case 12, Table I). Histamine flares placed on symmetrical areas of the dorsa of both feet revealed a much larger wheal and flare on the affected side. Following a lumbar sympathectomy the pain was completely relieved. The blood flow *decreased*. A shortening of the Achilles tendon was corrected by casts. The patient now works all day as a laundress.

Fig. 18. Case of M. O. with good history of Sudeck's atrophy 8 months after injury. The normal foot is on the right.

Late intractable case (Case 5, Table III)

M. B., 46 year old woman, fell over a rocking chair and twisted her ankle on getting up suddenly to answer the phone. She complained immediately of severe, continuous pain. At an emergency station a fracture of the fibula was diagnosed and a posterior molded splint was applied in an equinus position. Two weeks later another cast was applied in a somewhat better position. After 5 weeks in this cast, an adhesive strapping was applied to the ankle; she still had continuous pain and weight bearing was impossible. Because of the disproportion of the pain to the well healing fracture of the fibula the patient was suspected of malingering. A general anesthetic was applied and during the first stage moderate pressure was applied to the injured ankle. At the time she was first seen the foot was in marked equinus position, the skin was glossy and the ankle was swollen. There was an increased growth of hair on this area and the nails were ribbed. A four plus Wassermann reaction was obtained (Fig. 19a). Twelve blood flow determinations on each of the

Fig. 19. *A*, left, Photograph of M. B., 8 months after injury. The foot is discolored, swollen and in equinus position. *B*, The roentgenogram reveals diffuse bone atrophy.

extremities revealed very low basal readings (1—1.5 c.c. of blood per 100 grams of tissue) with only a slight increase of flow on the affected side. The volume of the injured limb was increased over that of its fellow. The affected extremity reacted very sluggishly to both a cold and hot water bath, the blood flows changing only very slightly.

X-ray films dating from 2 months after the injury revealed only a mild osteoporosis of the ankle and a marked localized rarefaction in the lower part of the fibula. The bone later became diffusely osteoporotic and took on a "ground glass" appearance which persists two years after the injury (Fig. 19b). She refused all therapy and has remained incapacitated to date. It is noteworthy that both hips and also her spine show a moderate osteoporosis. Whether the local atrophy accelerated the appearance of a senile osteoporosis is, of course, impossible to tell.

The type of injury, the faulty and insufficient immobilization, the mental attitude of the patient, and the unsympathetic attitude of her attendants all contributed to the outcome of this injury.

ANALYSIS OF STUDY

Many interesting and unsolved problems arise from the study of these reflex vasomotor disturbances. We shall discuss some of these in future publications. At present, we shall comment only on such findings as may contribute to a better understanding and an earlier treatment of Sudeck's atrophy.

In the first place, an early recognition of this syndrome seems important. In its milder forms, it is far from being rare. When a sprain or a blunt, minor injury does not show a normal course of events but is characterized by excessive pain, vasomotor phenomena and increased pulsations of peripheral vessels, an early reflex dystrophy should be suspected. As pain is purely a subjective phenomenon, the best diagnostic criterion is that of increased blood flow to the affected area. This can be substantiated by oscillometric curves, histamine flares, and skin temperature readings, as plethysmographic studies are obviously not available for clinical examinations. The truly functional, hysterical manifestations following trauma, which one of us (G. de T.) has had the opportunity to examine in 2 instances, are not associated with such vasomotor phenomena. It must be stressed, however, that the differentiation of an early Sudeck's atrophy from traumatic hysteria is often difficult, as the emotional status of many of these patients is often unstable. This we have discussed in an earlier communication. Of our present series, Case 18, E. H., has been repeatedly committed to an institution because of a severe psychoneurosis. Cases 27 and 28, seen quite early, exhibited other instances of autonomic instability, such as hypermotility of the intestinal tract and neurocirculatory asthenia. However, the "nervousness" and "excitability" of the late cases may just as well be the result of many months of suffering and not the cause of the syndrome, which most neurologists still maintain.

The osteoporosis, which some authors have placed in the foreground of their discussion is not as typical for this syndrome as one might suspect. It does not appear early, in fact, not

until 15 to 20 per cent calcium is lost from the bone. When it appears in its spotty form in the small bones of the hand and foot or in the lower metaphysis of the bones of the lower arm and leg, four to six weeks after the injury, it is rather characteristic. A diagnosis, then, can be made but only in conjunction with the clinical phenomena. In the later stages, when the bone atrophy is diffuse, a roentgenological differentiation from other forms of atrophy hardly seems possible (Fig. 18). Besides, undoubtedly an atrophy of disuse has been superimposed on the atrophy due to increased blood flow. These extremities are splinted by the physician or by the patient himself; they are not in use because of pain on motion or on weight bearing. For every case of late Sudeck's atrophy showing diffuse osteoporosis one can show many cases of severe bone atrophy after immobilization which are painless and do not show the characteristic features of this syndrome. We have already commented on the fact that minor, partial nerve injuries of peripheral nerves coursing superficially between the skin and bone may sometimes be detected as the initial stimulus for the vasomotor phenomena. Thus the dorsal interosseous nerve in Colles' fracture, the superficial radial and ulnar branches in twists and torsions of the wrist, the superficial peroneal and the posterior tibial and saphenous nerves in injuries to the ankle are often stretched, contused, or surrounded by a perineural hematoma. The diagnosis of such partial nerve injuries would probably be made more often if examination would be directed toward them in cases of severe pain following trauma.

There are still a large number of cases, however, in which such injuries are not demonstrable and in which the irritative nerve lesion must arise in the numerous receptors of the tendons or ligaments. The periarticular structures of small joints in the fingers are especially endowed with sensory end-organs. But if the continuous irritation of these can give rise to vasomotor phenomena, to trophic changes, to bone atrophy, why would not rheumatic polyarthritis bring about such changes. And truly, when one examines the cold, clammy hand of such a patient, with spindle shaped swelling around the small joints, with the marked atrophy of skin, contracture of tendons and joints with osteoporosis, the analogy between such a picture and a late stage of Sudeck's atrophy is striking. Another condition in which reflex vasomotor phenomena occur from a localizable nervous structure is the vascular syndrome caused by glomus tumors. The nonrecognition of a small subungual glomus tumor had led us to the diagnosis of Sudeck's atrophy following injury.

This was the case of T.C., a 60 year old machinist, who came to the Research and Educational Hospital on January 21, 1937, through the courtesy of Dr. Sumner L. Koch.

This patient complained of continuous severe pain in the right middle finger of 5 years' duration. In 1919 he had cut the tip of this finger which became infected. The scar became painful 10 years later and a spreading neuralgia developed first as far as the elbow, later ascending to shoulder and anterior chest wall. Squeezing the finger helped the pain. He kept a bucket of cold water next to his bed at night and plunged his hand in it for relief. The right hand was warmer than the left and there was a small linear, longitudinal scar at the tip of the right middle finger. The patient did not complain of any unusual sensitivity of the scar. Oscillometer readings showed a marked increase in oscillations on the affected side (Fig. 20).

A diagnosis of reflex traumatic vasodilatation was made. X-ray films revealed no osteoporosis. On March 23, 1937, an anterior cervicodorsal sympathectomy was done, resulting in complete dryness of the extremity and a Horner's syndrome. There was marked relief from the burning pain in the hand and the spreading neuralgia within 24 hours. However, it became apparent that the scar acted as a trigger zone and pressure upon it would elicit paroxysmal pain. Therefore, a week after the sympathectomy the scar was excised. A grayish nodule was observed within the fibrous scar, which was thought to be a neuroma. Histological examination, however, revealed it to be a glomus tumor. All symptoms disappeared.

Obviously the scar should have been excised before the sympathectomy, had one thought of a glomus tumor. In our experience, however, the removal of a neuroma in a scar or stump hardly ever gives lasting relief unless done quite early and usually aggravates the symptoms.

This case illustrates that a glomus tumor may become the origin of a chronic reflex

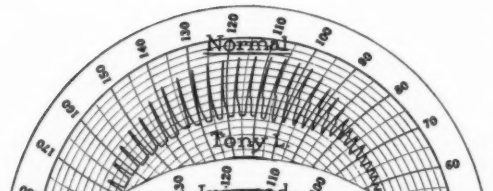


Fig. 20. Oscillometric curves of the normal and injured side. The individual pulse waves are higher on the injured side, but, in addition, the entire curve has shifted to the left. The diminution of spikes at 100 millimeters of mercury on the injured side and at 90 millimeters of mercury on the normal side indicates that the patient has vasodilatation with increased peripheral resistance.

vasodilatation. This has been observed and commented on by Stabins, Thornton, and Scott and is obviously another example of a vasodilator reflex in the extremities.

Blood gas studies performed by Dr. F. A. Hick 2 weeks after both operations revealed that venous blood from the affected arm had a 64.4 per cent saturation of oxygen, whereas, the venous oxygen saturation from the other arm was 58 per cent.

It is our contention that, when carefully examined, the early case of posttraumatic dystrophy will frequently reveal a source of continuous nerve stimuli which are then responsible for the sensory and vasomotor phenomena. If such patients' injured parts are adequately immobilized and rendered painless either by local infiltration of procaine or by sedatives and narcotics, the various short and long reflexes may not become established. It is a curious fact that severe fractures, which are promptly reduced and immobilized by closed or open reduction, never, and we say this advisedly, give rise to Sudeck's atrophy. It is the minor injuries, whose immobilization is seldom carried out very carefully, that are apt to be followed by this syndrome. The best prevention for Sudeck's atrophy seems the early and complete fixation of all injuries, without interfering with motion and permitting weight bearing, as Boehler recommended.

A paradoxical finding should finally be discussed. If vasodilatation, increased arterial pulsation, increased oscillometric readings, and increased blood flow can be demonstrated, especially in the early stages of Sudeck's atrophy, why should sympathetic block or sympathectomy be of help and bring about such relief as we have reported. There are several possible explanations which need to be examined:

First, it is possible that the painful stimuli, which give rise to vasodilatation, travel centripetally through the sympathetic fibers and ganglia. With the exception of Foerster and Leriche, all experimental and clinical investigators deny that any impulses from the extremities are carried by the sympathetic nervous system. It is true, however, that a sympathetic block promptly abolishes the causalgia, burning pain of this syndrome, without abolishing the vasodilatation. On the contrary it increases it.

This finding leads to another statement. The pain then is not due to vasodilatation, at least not the kind that block of sympathetic fibers produces; the pain causes vasodilatation.

The second possibility is that the reflex vasodilatation of Sudeck's atrophy is mediated by efferent vasodilators in the sympathetic nervous system. We have discussed the evidence that they do exist in man. However, if such a mechanism were operating, the pain would be unrelieved and vasodilatation abolished after sympathetic block, whereas, exactly the opposite is true.

The third possibility is that the vasodilator efferent fibers of the posterior root system, which are stimulated either directly or reflexly by the trauma, secrete a pain substance which is diffusible and would explain the peculiar types of hyperesthesia which are observed in Sudeck's atrophy. These fibers correspond to the "nocifensor system" of Sir Thomas Lewis. Sympathectomy, then, would not interfere with this nervous pathway, but by accelerating circulation, would eliminate or "wash out" the pain substance. This same mechanism would explain the fact that a herpes, seen in a causalgic hand, rapidly

healed after sympathectomy (15) and that shingles, which are certainly due to an irritation of the posterior root system, are rendered painless and made to heal after paravertebral block (our observations).

A final possibility should be mentioned. If the vasomotor disturbance in Sudeck's atrophy is such that an arteriolar dilatation exists together with capillary spasm (Fig. 20), then the pulse waves reaching the capillary bed may stimulate the sensory receptors and produce a throbbing pain with each pulse. Sympathectomy in such a case would release the venocapillary obstruction, and while blood flow would be further increased, it would not cause pain. There are certain observations to show that such a condition may exist in some patients. Thus, in a number of instances, the affected extremity is cold, clammy, sweaty, and shows the earmarks of sympathetic hyperactivity; it also feels cold to the patient, and yet the blood flow is increased. This is the case in hands of patients suffering from rheumatoid arthritis.

We have no data at present which would enable us to choose between the last two explanations. It may even be that both mechanisms may operate simultaneously or in successive stages. The hot, red extremity of a polyneuritis is sometimes followed by a cold, cyanotic stage and yet blood flow is still demonstrably increased. These problems await further investigation.

Whatever the explanation is, repeated sympathetic block in the earlier stages and sympathectomy in the later, more intractable cases have been of decided benefit. The procaine infiltrations can be followed by mild physical therapy which, if used alone, often aggravates the pain and muscle spasm. When organic changes are present, such as shortening of tendons, shrinking of joint capsules, ankylosis, and faulty position, they need orthopedic care for a full functional recovery after sympathectomy.

SUMMARY

Attention has been called to a not infrequent posttraumatic syndrome described by Sudeck in 1900. The diagnosis can be made by the early appearance of severe continuous pain, at first localized to the site of injury, later spreading proximally toward the root of the limb and sometimes even to the trunk and the contralateral extremity. The injury is usually comparatively mild and involves nerve trunks, blood vessels, or periarticular structures. Satisfactory immobilization, infiltration of the site of injury with procaine, or repeated paravertebral block with procaine may abort the progress of the disease. Next to the spreading neuralgia an increased blood flow to the affected extremity is demonstrable with the oscillometer, with histamine flares but more accurately with a water plethysmograph. Over 100 blood flow determinations have been made on 12 patients. A total of 33 patients suffering from early, intermediate, and late stages of the disease have been reported. The roentgen findings of a spotty, later diffuse, osteoporosis are helpful in corroborating the diagnosis but are not essential for the diagnosis. The treatment must fit the duration and severity of the syndrome and consists of repeated injections of procaine into the injured area, paravertebral sympathetic block, periarterial sympathectomy, and sympathetic ganglionectomy. Orthopedic correction may be needed for the late cases after the pain has been stopped

REFERENCES

1. ABRAMSON, D. I., ZAZEELA, H., and MARRUS, J. Am. Heart J., 1939, 17: 194.
2. ANDERSON, R., LIVINGSTON, W. K., and DOW, R. S. J. Neurophysiol., 1941, 4:427.
3. ANDREWS, E. Ann. Surg., 1927, 85: 116.
4. BAYLISS, W. M. The Vasomotor System. London: Longmans, Green & Co., 1923.
5. BISHOP, G. H., HEINBECKER, P., and O'LEARY, J. C. Am. J. Physiol., 1933, 106: 647.
6. BLACK, J. R., GHORMLEY, R. K., and CAMP, J. D. J. Am. M. Ass., 1941, 117: 2144.
7. BOEHLER, L. Treatment of Fractures. Translated by M. E. Steinberg. Vienna: W. Maudrick, 1929.
8. BROOKS, C. M. Am. J. Physiol., 1935, 106: 251.
9. BURN, J. H. Physiol. Rev., 1938, 18: 137.
10. CANNON, W. B., and ROSENBLUETH, A. Autonomic Neuroeffector Systems. New York: Macmillan Co., 1937.
11. DE TAKATS, G. Arch. Surg., 1937, 34: 939.
12. DE TAKATS, G. Trauma and peripheral vascular disease. In Brahdy, L. and Kahn, S.: Trauma and Disease. 2d ed. Philadelphia: Lea & Febiger, 1941.
13. DOLE, V. P., and MORISON, R. S. Am. J. Physiol., 1940, 130: 304.
14. FOERSTER, O. Die Leitungsbahnen des Schmerzgefühls und die chirurgische Behandlung der Schmerzzustände. Berlin: Urban & Schwarzenberg, 1927.

15. GASK, G. E., and ROSS, J. P. The Surgery of the Sympathetic Nervous System. 2d ed. Baltimore: Wm. Wood & Co., 1937.
16. HERRMANN, L. G., and CALDWELL, J. A. Am. J. Surg., 1941, 51: 630.
17. HESSER, F. H., LANGWORTHY, O. R., and KOLB, L. C. J. Neurophysiol., 1941, 4: 274.
18. HINSEY, J. C. Ann. Rev. Physiol., 1939, 1: 407.
19. HOMANS, J. Ann. Surg., 1941, 113: 932.
20. KARITZKY, B. Akute Gliedmassendystrophie in Ihrer Bedeutung fur die Behandlungsmassnahmen in der Unfallchirurgie, Hefte f. Unfallheilkunde 22. Berlin: F. C. Vogel, 1938.
21. KENNARD, M. Arch. Neur. Psychiat., 1935, 33: 537.
22. LERICHE, R. The Surgery of Pain. Translated and edited by Archibald Young. Baltimore: Williams & Wilkins Co., 1939.
23. LEWIS, T. Clin. Sci. 2: 373, 1936.
24. Idem. The Blood Vessels of the Human Skin and their Responses. London: Shaw & Sons, Ltd., 1927.
25. LEWIS T., and PICKERING, G. W. Heart, Lond., 1931, 16:33.
26. LOVÉN, C. Ber. Sächs, Ges. Wiss., 1866, 18: 85.
27. MIDDLETON, D. S., and BRUCE, J. Edinburgh M. J., 1934, 41: 49.
28. MOORE, R. M., and SINGLETON, A. O., JR. Am. J. Physiol., 1933, 104: 267.
29. PILCHER, R., WYATT, T. E., and CARNEY, H. M. Arch. Neur. Psychiat., 1941, 45: 321.
30. PINKSTON, J. O., and RIOCH, D. McK. Am. J. Physiol., 1938, 121: 49.
31. POMMER, G. Arch. klin. Chir., 1925, 136:1.
32. RANSON, S. W., and BILLINGSLEY, P. R. Ann. J. Physiol., 1916, 41: 85.
33. SCHUELE, F. Arch. orthop. Unfallchir., 1931, 38: 621.
34. STABINS, S. J., THORNTON, J. J., and SCOTT, W. J. M. J. Clin. Invest., 1937, 16: 685.
35. SUDECK, P. Arch. klin. Chir., 1900, 62: 147. Deut. Zschr. Chir., 1931, 234: 596. Arch. klin. Chir., 1938, 191: 110.
36. TINEL, J. Le système nerveux végetatif. Paris: Masson et Cie, 1937.
37. TOENNIES, J. F. J. Neurophysiol., 1938, 1: 378.
38. TURNER, M. Arch. klin. Chir., 1924, 128: 422.
39. WEISS, S., and ELLIS, C. B. J. Clin. Invest., 1930, 9: 17.

IMPROVED METHOD OF MEASURING THE POTENTIAL DIFFERENCE ACROSS THE HUMAN GASTRIC MEMBRANES AND ITS CLINICAL SIGNIFICANCE

A Preliminary Report

EDMUND N. GOODMAN, M.D., New York, New York

IN 1936 Adair and Goodman reported a method of measuring the potential difference across the stomach membranes in an effort to quantitate the state of vagotonia often observed in cases of peptic ulcer. In 1937, in collaboration with J. P. Quigley and Sir Joseph Barcroft, certain factors modifying the potential were elaborated. It was concluded that the gastric potential is not dependent on the hydrogen-ion concentration of gastric contents nor upon the well known types of gastric activity—that is, the secretion of acid, mucin, or pepsin—nor upon hunger contractions, digestive contractions, or other types of motor activity, nor upon the diffusion of ions or nonelectrolytes. The potential difference within the lumen of the stomach or of a gastric Pavlov pouch is negative with respect to the abraded skin and during fasting periods the potential difference is relatively constant. The potentials of the cardia and pylorus differ about 5 millivolts in magnitude but generally change in the same direction. The potential difference is lowered by contact of milk, emulsified fats, alcohol, or dextrose with the gastric surface, but in the case of the latter two not by their introduction in the blood stream. It was felt that this gastric potential difference depends on an undetermined type of activity, apparently of the gastric mucosa, and that when altered by contact of the mucosa with effective substances, the influence may be transmitted to gastric tissue not in contact with the excitant.

In 1939 the method was modified and applied to humans. Through the suggestions and collaboration of Dr. Kenneth Cole of the department of physiology of the College of Physicians and Surgeons of Columbia University the type of electrodes was changed. We employ liquid junctions which are easily renewable by washing and the electrodes do not make direct contact with living tissue. The potential differences are balanced by a recording potentiometer. These potentials are then independent of resistance changes and electrode polarization effects in the system measured.

Figure 1 illustrates the construction of the electrodes. The insulated copper wires are attached to a Leeds and Northrop special micromax recorder. The external resistance of the circuit is in the range of 50,000 to 100,000 ohms. The recorder has a range of 0 to 100 millivolts with an accuracy of 1 per cent with an external circuit resistance of 50,000 ohms and an accuracy of 1 to 2 per cent with an external circuit resistance between 75,000 and 100,000 ohms. There is also a special shunt whereby the electrodes may be calibrated against each other. The recorder is set to run at 1.4 centimeters in 5 minutes.

Figure 2 shows the calibration of one electrode against another. This is done by placing the side arm tubes of the electrodes in normal saline and 1/10 normal hydrochloric acid, and running the machine at a known resistance recorded on the shunt. This shows that there is practically no difference between the two electrodes as the shunt resistance and recorded voltage is essentially the same.

Figure 3 shows the actual setup on a patient. The electrodes are prepared for use and applied to the patient in the following manner:

The T-tube pyrex glass tubing, in one end of which is sealed a piece of platinum, is thor-

From the Department of Surgery, Columbia University and the Surgical Service, Presbyterian Hospital, New York.
Aided by a grant from the Commonwealth Fund.

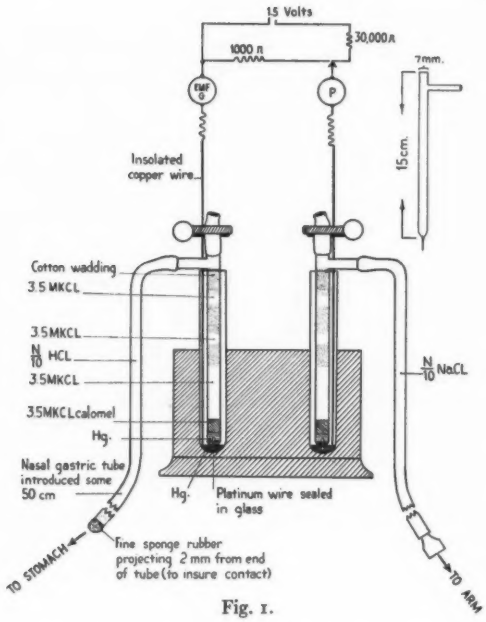


Fig. 1.

oughly cleansed and dried. It is then filled as shown with a small quantity of clean, dry mercury to cover the protruding platinum wire and layered on this is a small quantity of calomel mixed with 3.5 molar potassium chloride. The tube to the T-joint is then filled with 3.5 molar potassium chloride and 3 pieces of cotton wadding are introduced at convenient distances. This prevents dispersion of the electrolytes and after the electrodes have been used some 3 to 5 times, the upper 2 cottons are removed and fresh 3.5 molar potassium chloride introduced. Some 20 minutes before use on the patient the electrodes are further prepared by attaching the side arm of one to a rubber tube to be used for the arm lead, and filling this with 1/10 normal sodium chloride solution by aspiration with an asepto syringe. The fluid remains in the tube since one end is clamped. The other electrode is similarly prepared. However, the nasal gastric tube is more easily filled by injecting the 1/10 normal hydrochloric acid solution into the nasal gastric tube. After both have been filled the apparatus is allowed to stand 20 minutes before use.

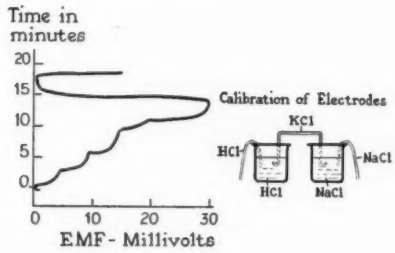


Fig. 2.

The test is performed on a fasting patient with an empty stomach. If there is reason to believe that there is gastric retention, the stomach must be lavaged clean before the test is performed. Supper is allowed the evening previously but no further food or liquid other than water until the following morning when the test is usually run between 7:30 and 8:00 a.m. The routine employed has been:

1. Nasal application of 2 per cent cocaine in spray is made.

2. The right forearm ventral surface is slightly abraded with a needle and a rubber cuff containing a glass cup is applied over the abraded area, and the cup is filled with 1/10 normal sodium chloride solution. The filled nasal gastric tube attached to the side arm of the electrode is then introduced through the nose into the stomach, some 50 to 56 centimeters. The arm lead is then attached to the glass cup and the record is started.

3. An immediate response is recorded and this is allowed to progress until either a steady baseline is obtained or until it is apparent that the response is assuming a definite pattern. This usually requires some 10 to 15 minutes. When the latter occurs, the patient drinks 150 to 200 cubic centimeters of milk and the change in potential is noted for another 10 to 15 minutes.

Eight to 10 runs are performed on a set of electrodes.

EXPERIMENTAL DATA

The following is a preliminary report of data concerning the different electrical potential responses evoked by milk in the various gastrointestinal lesions. In the preceding experimental work, it was shown that milk

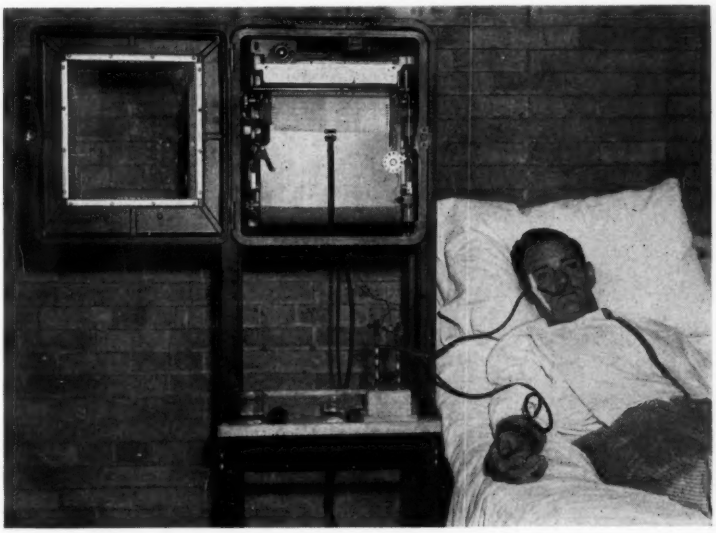


Fig. 3.

altered the potential in a fairly uniform pattern in the dog and sheep. By the use of the response to milk in humans, it was soon apparent that so called normal humans likewise had a potential difference of a fairly constant character and magnitude. With this phenomenon the stomach is being studied in various diseased conditions to ascertain whether it reacts in different and characteristic fashions. The following data are the empirical correlations of the standard test response to milk with known disease found at operation or through clinical course.

Before proceeding with the various lesions, certain data are pertinent showing that (1) the milk response occurs only when there is contact with gastric mucosa and will not take place *in vitro*, (2) the response to milk seems to be specific for the stomach.

The potential difference between the contents of the two beakers is noted. Milk is added to the beaker containing gastric juice. No change in the potential difference occurs (Fig. 4).

Mr. L., a patient suffering from cardiospasm some 40 years, 4 years ago required a gastrostomy for feeding purposes. With the gastric tube placed in the stomach through the gastrostomy opening milk in the esophagus taken by mouth fails to alter the potential difference in the stomach. However, immediately the patient forces milk through the esophagus into the stomach, a response occurs (Fig. 5).

Mrs. H. had a total gastrectomy some 3 weeks before the curve illustrated in Figure 6 was made. With the nasal tube through the anastomosis in the jejunum, milk fails to evoke a response.

The next three graphs are introduced to show that the bizarre patterns sometimes obtained in the gastric lesions are not necessarily artefacts due to changes in the electrodes.

Figure 7 shows a potential difference response in a normal subject, performed on March 4, 1941. Figure 8 shows the potential difference response of a patient having intermittent gastric dilatation and hemorrhage due to malnutrition and cachexia. Figure 9 shows the potential difference response of the same patient as in Figure 7, but on March 7, 1941 using the same electrode as in Figure 8 and thereby demonstrating that the bizarre response in Figure 8 is not due to electrode phenomenon.

Figures 10, 11, 12, 13 are representative curves in a series of cases of the various lesions to be presented. These were compiled from the first 150 cases done with the present setup.

The following is the elaboration of characteristics of response in the various groups of lesions.

No known disease (Fig. 10). The characteristics of 24 of 25 cases may be summarized as having: (a) a steady baseline, (b) an immediate response to milk, (c) a smooth curve

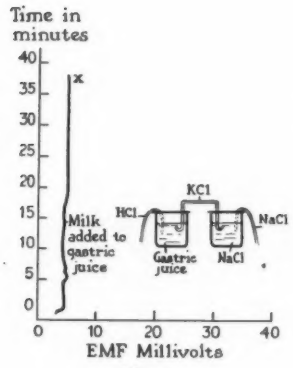


Fig. 4.

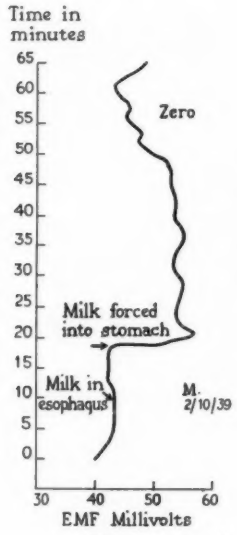


Fig. 5.

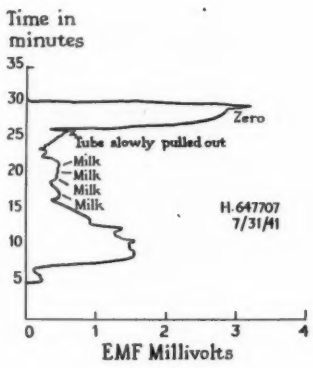


Fig. 6.

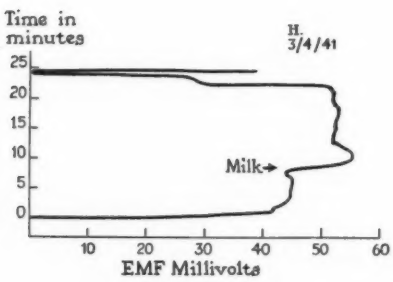


Fig. 7.

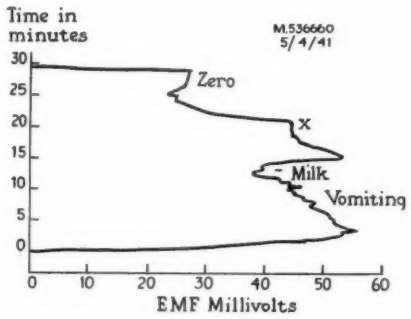


Fig. 8.

as a response, (d) a range of between 40 and 60 millivolts, (e) a highest rise of 11 millivolts and the lowest of 3½ millivolts, (f) a sustained rise averaging 5 minutes, the shortest 4 minutes, the longest 15 minutes.

One subject whose curve is not shown presented an irregular baseline and an irregular sustained rise after an immediate reponse to milk. This subject was an interne at the time under considerable distress and was later shown to have considerable pylorospasm.

No intrinsic disease of the stomach—known disease elsewhere. These 10 cases represented carcinoma of the cecum, adenofibroma of the breast, carcinoma of the head of the pancreas, carcinoma of the sigmoid and a ventral hernia, also retroperitoneal sarcoma, metastasis to spine—primary carcinoma, site unknown, gall-bladder disease, etc.

All of this group showed: (a) an immediate response to milk, (b) a steady baseline, (c) a smooth curve response, (d) a range of between 35 and 60 millivolts, (e) a highest rise of 14 millivolts and lowest rise of 3 millivolts, (f) a sustained rise averaging 5 minutes, the lowest being 2½ minutes. The characteristics of this group are similar to the normals.

Duodenal ulcer (Fig. 11). The verification of diagnosis in this group of 25 cases was confirmed either by repeated x-ray examination

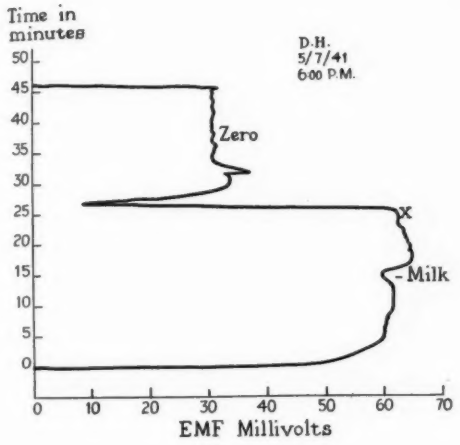


Fig. 9.

or by pathological examination after operation. They include 3 patients who had had a previous gastroenterostomy who were found to have duodenal and jejunal ulcers. One patient in the group in addition to having a duodenal ulcer was found to have unsuspected chronic myeloid leucemia. The tests were made before operation.

The characteristics of this series are: (1) a steady baseline when there is no associated gastric pathology, (2) an immediate response to milk, (3) a smooth curve, (4) a highest response of 11 millivolts, lowest of 2¼ millivolts, (5) a shortest sustained rise of 5 minutes and longest of 10 minutes, (6) a voltage ranging between 35 millivolts and 60 millivolts. These characteristics were noted in all of the 25 cases.

Gastric ulcer (Fig. 12). The diagnosis in this group of 30 cases was confirmed either by operation or repeated gastrointestinal series with follow-up. Twenty patients were operated upon, partial gastrectomy being done. The 10 remaining patients had repeated gastrointestinal x-ray films and have been followed for an average of 7 months. The tests were made before operation.

An illustrative case is presented.

A man aged 56 years had a 6 months' history of insidious anorexia and acute history of 12 hours of vomiting (Fig. 14). Two gastrointestinal series by our senior roentgenologist were interpreted as carcinoma of the prepyloric region. The patient had an average free hydrochloric acid. At operation there was found a sharply defined, hard lesion involving the pylorus and prepyloric region with a white zone on the corresponding serosa. There was one enlarged lymph node. However, microscopic section revealed a gastric ulcer with hypertrophy of the pyloric muscle and no carcinoma. A follow-up of 6 months shows the patient in excellent condition.

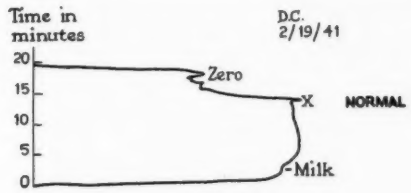


Fig. 10.

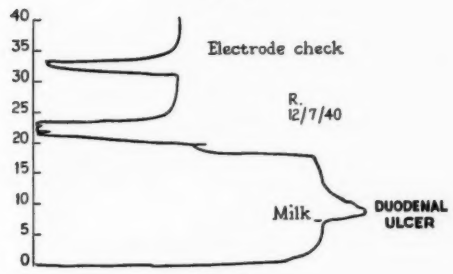


Fig. 11.

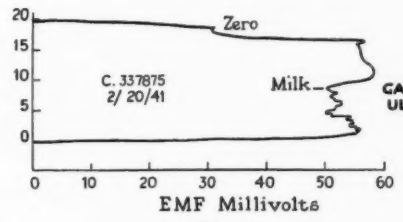


Fig. 12.

The characteristics of this group are: (1) the baseline tends to be irregular with the interposition of short, fast deflections of the galvanometer; (2) there is an immediate response to milk; (3) the curve may not be a smooth curve, the same short deflections being interposed on the curve. The response to milk is greater in millivolts than any of the irregularities of the baseline. The highest response was 13½ millivolts and the lowest 2¼ millivolts; (4) the average rise was 5 minutes and the longest, 15 minutes. This group differs from

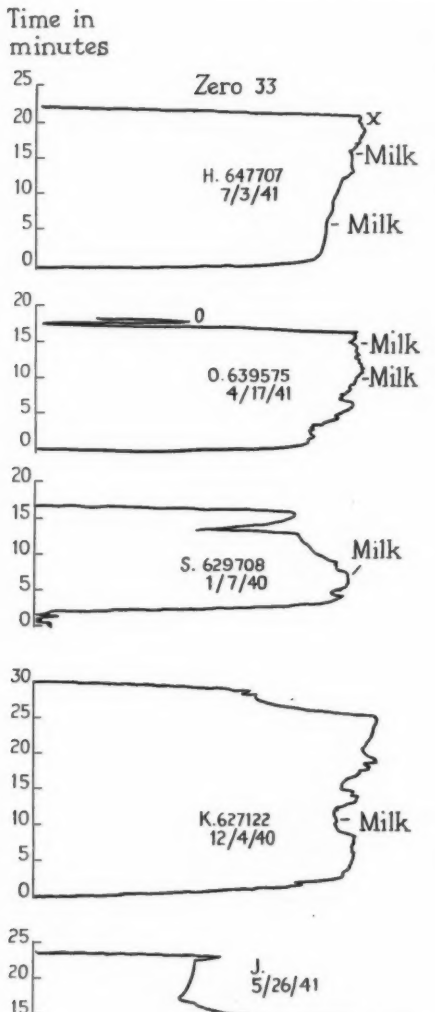


Fig. 13.

the duodenal ulcer group and from the normal by the interposition of the short fast deflections. In this group of 30 cases, 26 of the 30 exhibited the characteristics of response elaborated. Of the 4 cases which showed different characteristics, 1 had a rise in potential similar to the above but the baseline had been falling previous to the introduction of milk and the response in potential difference was not higher than the highest point on the baseline although the type of response fell into the same characteristics as the general group. This case showed duodenal and gastric ulcers with papillomatous proliferation of Brunner's glands projecting into the pylorus and with polypoid hyperplastic lymphoid gastritis of the antrum. Two cases showed a bizarre, irregular baseline with a bizarre, irregular response to milk. One was a case of recent gastric perforation with a prepyloric mass which required a secondary gastroenterostomy for obstruction. The lesion was thought to be an ulcer because of the favorable follow-up of some 3 months. The other case presented an unusual picture of multiple, shallow ulcers surrounded by inflammatory cells, the ulcers being mainly in the mucosa and submucosa of the proximal half of the stomach with 2 suspicious bowl-shaped ulcers with sloping margins in the distal half of the stomach. The fourth case was a man with a gastric ulcer which grossly was suspicious of cancer but turned out to be nonmalignant on section. This man had 2 curves, one of which was characteristic of the response noted above, the other of which, however, failed to give a response to milk though the baseline was falling before milk was given.

This group of 30 cases is of interest because from a clinical, roentgenological, and gastroscopic opinion the diagnosis was most difficult. The x-ray findings in this group are summarized by Dr. Paul Swenson of the department of roentgenology. This group of 30 cases averaged over 3 gastrointestinal x-ray examinations.

Six were diagnosed as duodenal ulcers, 8 as benign gastric ulcers, 9 as suspicious of cancer, and 7 were definitely diagnosed as cancer. Dr. Swenson states:

"This series that we have studied includes 30 cases of peptic ulcer in which the differentiation between ulcer and cancer was not certain. In all but a few, the question of neoplasm

had been raised at some time during the series of examinations. There were certain roentgen criteria which made the examination favor one more than the other. There were 8 cases considered benign because the character of the crater shadow and its definite reaction to dietary regimen. However, in 16 cases the radiologist or the several roentgenologists felt that the differentiation between ulcer and cancer simply could not be made from the x-ray findings alone. The findings upon which this suspicion of neoplasm was based were: (1) the failure of the crater to decrease in size upon therapy; (2) the lack of symptoms; (3) a relatively large area of inflexibility around the ulcer crater suggesting either malignant infiltration of the wall or a large area of inflammatory induration, the two being indistinguishable.

"On the side of benignity the suggestive roentgen evidence was: (1) the definite diminution in size of the crater under therapy (not wholly a reliable sign); (2) the intermittent relaxation of areas of constriction which proves that these were areas of spasm rather than infiltration; (3) the peristaltic activity within the area of supposed involvement.

"The amount of retention in the stomach had little to offer either way as far as the differentiation of carcinoma from ulcer was concerned. It is of interest that the examiner definitely favored neoplasm in a fairly large number of cases. The percentage of these cases in which neoplasm was definitely favored would, I believe, be about the same as that in a similar series in a carcinoma group in which the same criteria come into use. In other words, there is a certain group in which the radiologists simply cannot be certain without a number of examinations during therapy to watch for any change."

The gastroscopists have been consulted in 6 of these 30 cases; 3 were diagnosed as cancer. Therefore the gastroscopist is unable to be more specific than the radiologist.

Other gastric diseases (Fig. 15). This miscellaneous group of 10 cases is included although there is an insufficient number to indicate that these conditions show a different response from the cancer group. These cases include hypertrophic gastritis, atrophic gastritis with

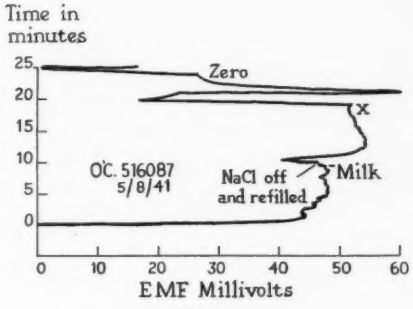


Fig. 14.

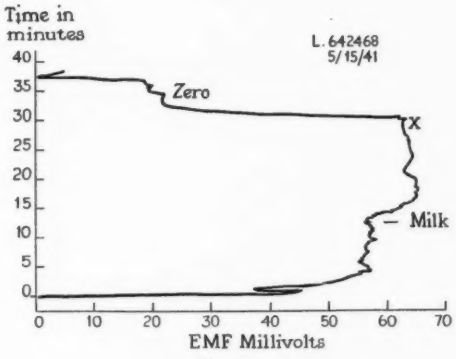


Fig. 15.

achlorhydria, duodenitis with herniation of gastric mucosa into antrum, pylorospasm with duodenal ulcer and associated gastritis, and cases of gastric symptoms but with entirely negative laboratory findings. A case of gastritis is shown in Figure 15. This series revealed a tendency toward an unsteady baseline and immediate response to milk, the curve of which tended to have the interposition of short, irregular deflections, a range between 40 to 60 millivolts, the shortest sustained rise 5 minutes and the longest 30 minutes, the rise in all cases averaging 5 to 16 millivolts. This group all exhibited the characteristics of the gastric ulcer pattern in which it is noteworthy that short, rapid deflections occur.

Carcinoma. This group of 46 cases, though hardly presenting a sufficient number of cases, is worthy of a preliminary report in that it appears that the response to milk in the electric potential difference in cases of carcinoma is different from the response of other lesions.

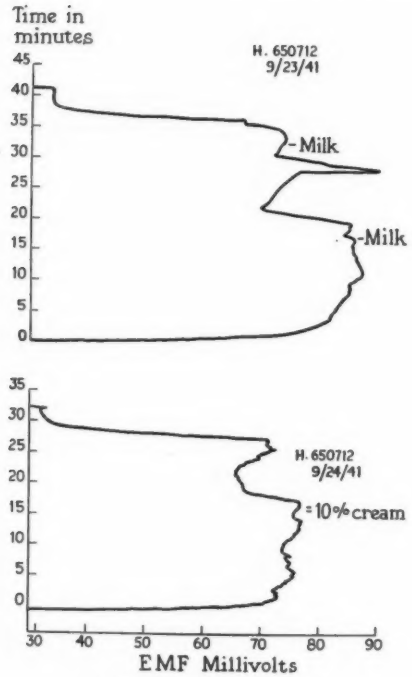


Fig. 16. Before operation.

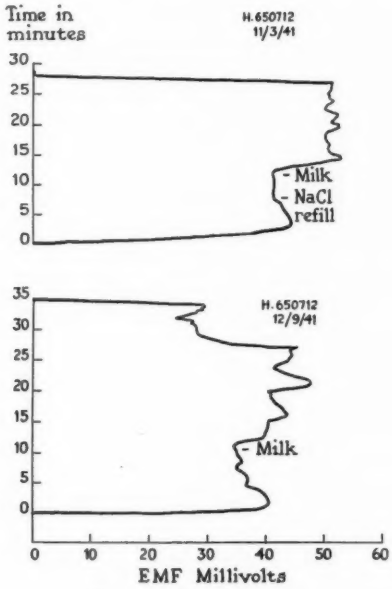


Fig. 17. After operation.

All of the cases but 1 have been verified at operation with microscopic sections.

Figure 13 shows 5 cases of cancer, the responses of which may be characterized by a steady rise in the electric potential with the superimposition of short, fast deflections and in which there is no marked response to milk. In the second group there is a general rise in the potential difference on which the same short, fast deflections occur and there is no response to milk. In the third group there is a negative response to milk. The fourth group showed irregular, box-like pattern of the baseline with the interposition of the short, fast deflections and with the same pattern being repeated after the introduction of milk. A fifth type—there are 4 cases in this series—shows an irregular baseline of these same characteristics and after on introduction of milk an immediate spike-like rise and fall of the potential, lasting about 2 minutes. All 4 of these cases have a very advanced malignancy with multiple gross metastases.

Two further cases are given in detail to indicate the probable usefulness of this method:

A 56 year old porter (Figs. 16 and 17) had epigastric pain, vomiting, and weight loss over a 2 year period but much worse for 2 weeks before admission. He had a known duodenal ulcer history of 25 years' standing with symptoms of intermittent hemorrhage and pain. On admission, the gastrointestinal series revealed 100 per cent gastric retention and only slight free hydrochloric acid. There was not a great deal of improvement on lavages, and a gastroenterostomy under local anesthesia was performed, revealing a crooked, scarred area in the prepyloric region. This did not have the gross appearance of cancer. However, the patient failed to improve after operation, at which time the x-ray examination revealed obstruction of the gastroenterostomy and the inability to force barium through the pylorus. At this time the roentgenologist raised the question of malignant neoplasm of the antrum. The patient was reoperated upon on September 25, 1941, at which time there was described edema of the distal limb of the gastroenterostomy and the same puckered, innocuous-looking scar in the antrum. A subtotal gastrectomy was performed with dissolution of the gastroenterostomy. Microscopic section revealed a superficial spreading carcinoma in the region of the old gastric ulcer, the tumor at only one point penetrating to the adjacent muscularis. There were metastases in 3 inferior gastric lymph nodes. There was no real obstruction at the pylorus.

In this case it was felt reasonable to assume that the lesion of the stomach had been successfully extirpated. The potential difference response before and after operation is shown, indicating an entirely different response postoperatively.

A second case (Fig. 18) is given of a 47 year old colored woman who in 1924 had a cholecystectomy. For 2 years previous to admission she complained of abdominal pain, nausea, anorexia, and weight loss. Six months before admission these symptoms became worse. X-ray examination revealed an obstruction near the pylorus with tenderness in this region and the antrum failed to contract in systole. The patient had a normal free hydrochloric acid. At operation a subtotal gastrectomy was performed for a stenotic ulcer at the pyloric junction penetrating along the posterior wall on to the pancreas. Grossly this had none of the characteristics of cancer. However, microscopic section revealed a poorly differentiated and diffusely infiltrating carcinoma developing on the base of an old ulcer with metastases to 4 lesser curve lymph nodes. The resection seemed to have been through tumor tissue. The electric potential difference response before and after operation is shown and the curves seem to be identical in characteristics.

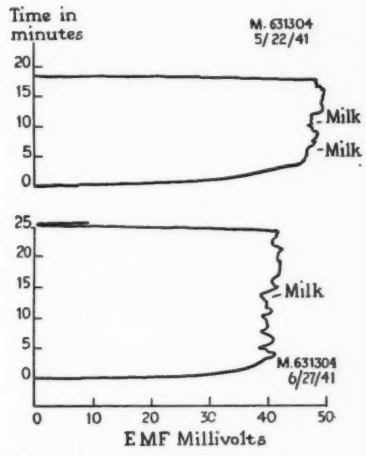


Fig. 18. Above, Before operation; below, after.

In this series of 46 cases there were 5 cases which differed in characteristics from the others. One, a man with a very advanced lesion presenting a mass in the abdomen and an enlarged liver, had an irregular baseline with a sharp response to milk. There were 2 patients who on different preoperative tests revealed curves, which at one time were characteristic of the cancer group and at another time of the ulcer group. The fourth case was a man with an advanced lesion in which there was a response though this response was not as high as the former baseline. The fifth case was a young man with extensive lesion penetrating through and invading the liver, in which there was an irregular baseline, a rise to milk with resumption of the same irregular baseline.

SUMMARY

A method is presented for measuring the potential difference across the human gastric membranes. This method is noteworthy in that the electrode system makes no direct contact with the skin or stomach.

A standard test was evolved using the potential difference response to milk as the control measurement. Data are presented on the various lesions indicating a different response to milk in the corresponding lesions.

CONCLUSIONS

Though the gathered data are suggestive, there is not sufficient material for conclusive deductions.

More accurate diagnoses on these types of cases should be made using this method in conjunction with roentgenological interpretation and gastroscopy.

The method appears useful in differentiating gastric carcinoma from other gastric lesions and this phenomenon seems more consistent the earlier the gastric carcinoma, both from time of onset of symptoms and from the extent of spread. The short, fast deflections mentioned appear only in gastric lesions and the true form and characteristics of the short, fast deflections which may have characteristics for each group will have to be investigated with a more sensitive and faster recording apparatus. There are many phases of the problems under consideration and experimentation, notably: (1) the responses obtained after gastrectomy, (2) the sources of the potential difference, (3) the different electrical properties of cells in correlation to different lesions, (4) the nature of the short, fast deflections.

REFERENCES

1. ADAIR, G. S., and GOODMAN, E. N. Proc. Physiol. Soc., J. Physiol., 1936, 87: 35–36.
2. BEUTNER, R. Physical Chemistry of Living Tissues. Baltimore: The Williams & Wilkins Co., 1933.
3. BIEDERMANN, WILHELM. Elektrophysiologie. P. 425. Jena: Fischer, 1895.
4. BOHLEN, F. Pflügers Arch., 1894, 57: 97–122.
5. BURR, H. S., SMITH, G. M., and STRONG, L. C. Am. J. Cancer, 1938, 32: 240–248.
6. CHAVOGETS, V. Y. Vrach. delo, 1935, 18: 367–368.
7. FLOYD, W. F., and KEELE, C. A. Proc. Physiol. Soc., J. Physiol., 1936, 86: 23–25.
8. HASAMA, B. I. Pflügers Arch., 1935, 236: 545–553.
9. QUIGLEY, J. P., BARCROFT, J., ADAIR, G. S., and GOODMAN, E. N. Am. J. Physiol., 1937, 119: 763.
10. ROSENTHAL, I. Arch. Anat. Physiol., 1865, 32: 301–316.
11. Idem. Fortschr. phys., 1870, p. 545.
12. SARRE, H. Zschr. Biol., 1930, 91: 37; 1930, 92: 495; 1934, 95: 135–145.
13. TITAEV, A. A. Bull. bio. méd. exp. U. R. S. S., 1940, 9: 96–99.
14. Ibid., 1938, vol. 5.
15. Ibid., 1938, vol. 6.
16. V. TSCHERMAK, A. Pflügers Arch., 1919, 175: 165; 1930, 224: 356.
17. VEIT, H. Zschr. ges. exp. Med., 1927, 56: 61–75.
18. WALLER, A. D. Arch. sc. biol., St. Petersburg, 1904, 11: 222.
19. WEITZ, W., and VOLLERS, W. Zschr. klin. Med., 1925, 102: 141.
20. Idem. Zschr. ges. exp. Med., 1925, 47: 42.

CLOTHESPIN OR INCLUSION GRAFT FOR SPONDYLOLISTHESIS OR LAMINAL DEFECTS OF THE LUMBAR SPINE

DAVID M. BOSWORTH, M D., F.A.C.S., New York, New York

BECAUSE of our dissatisfaction with the results of spinal grafts in spondylolisthesis and the difficulty of satisfactorily bridging laminal defects with grafts of previous types, we attempted to fasten tibial grafts in place with vitallium screws. This proved unsuccessful. The screws worked loose and contact could not be maintained. Absorption of massive portions of the grafts then took place. It occurred to us that if a graft could be devised which by its shape would be rigidly maintained in place in the lumbar spine without other method of fixation, better results in these two conditions could be secured. At first we designed an inclusion graft to place over the spinous processes of vertebrae which we desired to fuse. The patient was placed in a position of hyperextension before the graft was applied (Fig. 1, a). Such a graft was used successfully in a case of spondylolisthesis in conjunction with chips of bone removed from the ilium and placed beneath the graft. Though this case progressed to an entirely satisfactory conclusion and solid ankylosis, the technical difficulty of making an inclusion graft, and its weakness when made, forced us to use a stronger substitute. We therefore devised the double clothespin graft (Fig. 1, b). In this the intervening spinous processes were removed between the vertebrae to be ankylosed and the two ends of the graft so fashioned as to include the spinous processes of the end vertebrae of the fusion area. It was found at operation that besides strength another favorable factor was secured by the double clothespin graft. With the patient in a flexed position, so as to distract the spinous processes between which the graft was to be placed, a leverage action was secured by the abutment of the spinous processes on the ends of the graft when the patient was straightened out. There was actually distraction of the articular facets, opening of the intervertebral foramina, and separation of the vertebral bodies (Fig. 2). This gave theoretical reduction, at least, of any pressure on nerve roots passing out through the intervertebral foramina. Such actual distraction of the vertebral bodies included in the grafted area has been observed. Possibility of further displacement of spondylolisthesis by distraction has been watched for but not observed. On the other hand, replacement of spondylolisthesis by the distraction has likewise not been seen. Fixed continuity of transplanted bone with the grafted area has always been present.

We have used 1 inclusion graft, 16 double clothespin grafts, and 1 single clothespin graft. In the 18 operations done, there were 6 cases of mild shock, 2 of moderate shock, and in 1 patient death occurred 2 days after operation. The ages of the patients ranged between 18 and 55 years. Seven patients were women. All were of the white race, most of them Americans, and all walks of life were represented. The operation was performed for spondylolisthesis in 6 instances (Fig. 3), 5 of them being of the 5th lumbar and the other, a posttraumatic fracture spondylolisthesis, of the 4th lumbar. In all there were 12 patients in whom a double clothespin graft was placed in association with the creation of laminal defects. In 11 patients the graft was used to reinforce a defect due to posterior herniation of the nucleus pulposus and accompanied partial or complete laminectomy (Fig. 4). In 1 instance it was used to bridge a massive defect of several vertebrae necessitated by exploration and removal of a cord tumor in which articular facets were removed and stability of a long section of the lumbar spine lost. This exploration and laminectomy were performed by another surgeon, the graft being laid by us.

In 2 instances, laminectomy defect had been created at previous operations. All other lami-

From the Orthopedic Service of St. Luke's Hospital, New York.

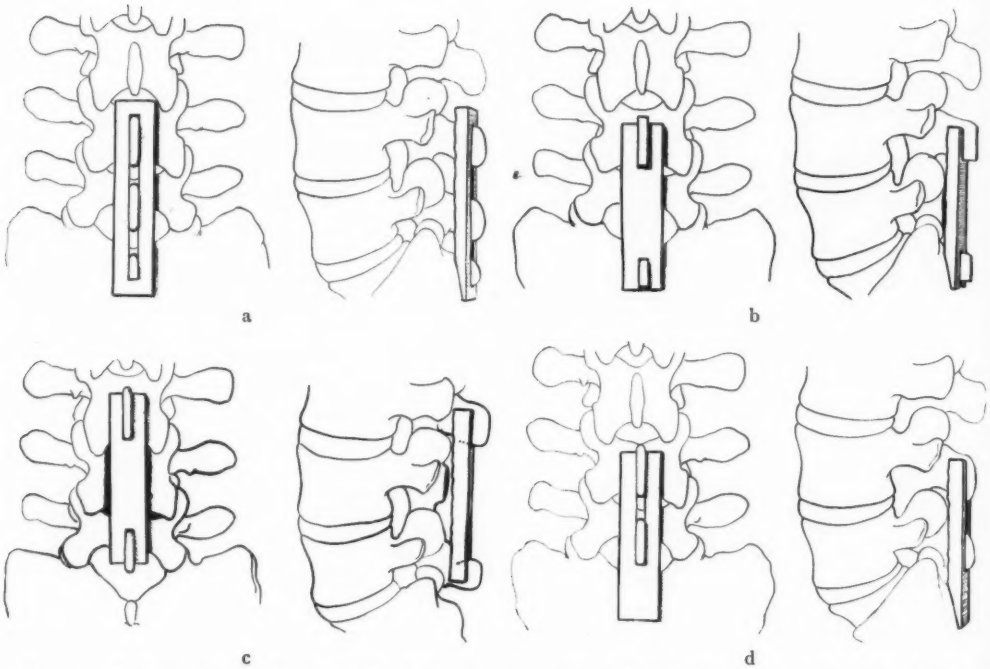


Fig. 1. a, Inclusion type graft. b, Double clothespin graft set between spinous processes of 4th lumbar and 1st sacral. c, Double clothespin graft set between spinous processes of the 3d lumbar to 5th lumbar, bridging laminectomy defect of 4th lumbar. Note undercutting of spinous processes. d, Single clothespin graft as used from spinous processes to previous spine fusion below or to sacral surface which lacks spinous processes.

nal defects were created and accompanied at the same operation with grafts of this type.

The length of time of operation varied from 60 minutes in spondylolisthesis to 2 hours or more in laminectomy cases, depending upon the necessity of search for or exposure and removal of any pathological condition present in the latter. Operative time was markedly reduced when two capable teams were associated, one in exposing the spine and the other in securing the tibial graft.

The clothespin grafts were reinforced with iliac chips on 11 occasions and with additional tibial bone on 3 occasions. In one instance the slot in the lower end of a graft was cut too narrow for the spinous process. As it was being driven on, one of the prongs broke off. Despite this, good stability remained at operation. Subsequently, the lower end displaced laterally but maintained contact with the spinous process and sacrum. Review of postoperative x-ray films showed that a possible fracture of the upper prong of another graft may have occurred, this without displacement of either the main graft fragment or the prong. No fracture of the main body of the graft has occurred in any of the cases to date. Immediate stability of all grafts, both inclusion and clothespin, was excellent. Final stability of all grafts has remained good to date. In 10 of the 18 cases, jacket cast was applied immediately after operation to prevent the possibility of the patient being drawn into the flexed position and of displacement of the graft. Since, however, in 8 patients no cast was applied after operation and there was no displacement of the grafts, it is believed by the author that a jacket cast is an unnecessary detail.

All wounds with the exception of 1 healed *per primam*. One other spinal wound broke down 3 weeks after suture removal. In both in-

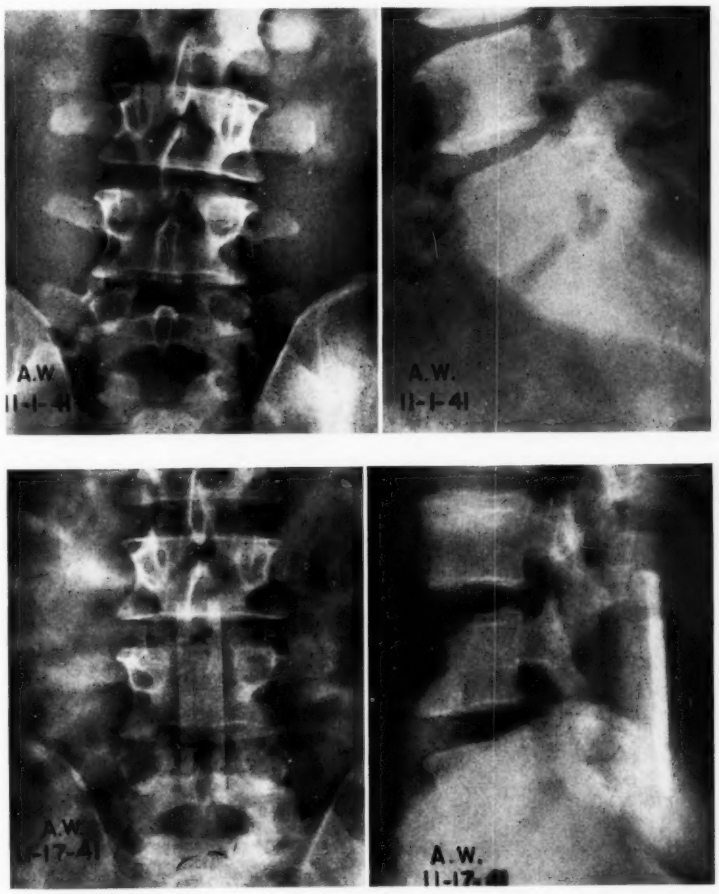


Fig. 2. A. W. By contrasting the spaces of the intervertebral disc and the intervertebral foramina in the upper pictures with those below, it can be seen that actual distraction of the posterior and anterior elements has occurred.

stances, a silk suture has been extruded, and in 1 healing has taken place. The probabilities are that other deep silk sutures will have to be removed from the unhealed wound. All tibial wounds healed *per primam*, but in 1 instance, in which sulfathiazol was used, the wound reopened 5 weeks after operation, drained a few days, and then healed. The average period before the patient became ambulatory has been 21 to 24 days after operation, though 1 patient became ambulatory after 9 days and 2 others after 18 days. The local condition of the site of removal of tibial bone will control the return to weightbearing. Absorption of graft or spinous process due to pressure contact has not been observed in any instance.

The extent of the graft has been from the 3d lumbar to the 1st sacral, 8 times; the 4th lumbar to 1st sacral, 4 times; the 3d lumbar to 5th lumbar, twice; the 4th lumbar to 2d sacral, twice; and the 2d lumbar to 5th lumbar, once. One additional graft was placed across 5 vertebrae for defect caused by exploration and removal of spinal cord tumor by another surgeon.

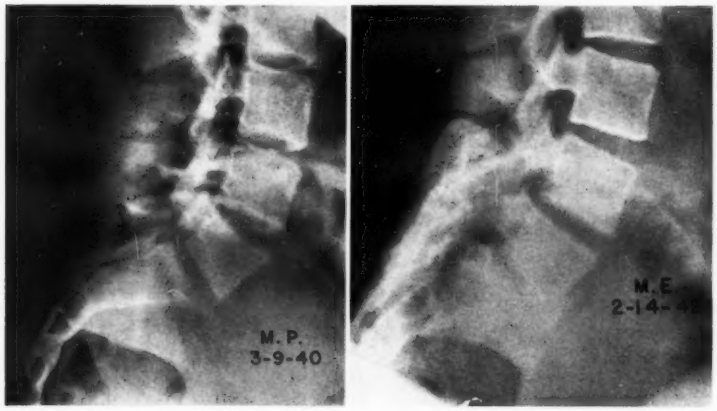


Fig. 3. M. P. Massive arthrodesis of the 4th lumbar to 4th sacral for spondylolisthesis.

DESCRIPTION AND MINUTIAE OF OPERATIVE PROCEDURE

The clothespin graft consists of an abutment of bone between two given spinous processes held in place by prolongations from either corner of the ends of the bony graft, the latter giving the appearance of a clothespin (Fig. 5). In the inclusion graft, the reverse effect is secured with a loop of bone in the form of a graft enclosing the spinous processes. As we do not favor the use of the latter type of graft, we shall not continue its description. In a typical single or double clothespin graft, the posterior elements of the laminal arches are denuded and decorticated except for the spinous processes at either end of the area to be grafted. These are retained. Occasionally in a severe spondylolisthesis, the posterior arch of the displaced vertebra will have been driven back so that it partially overrides the arch of the vertebra above and below. Such an arch may be removed to allow for the application of the straight clothespin graft without leaving much of a defect in the continuity of the

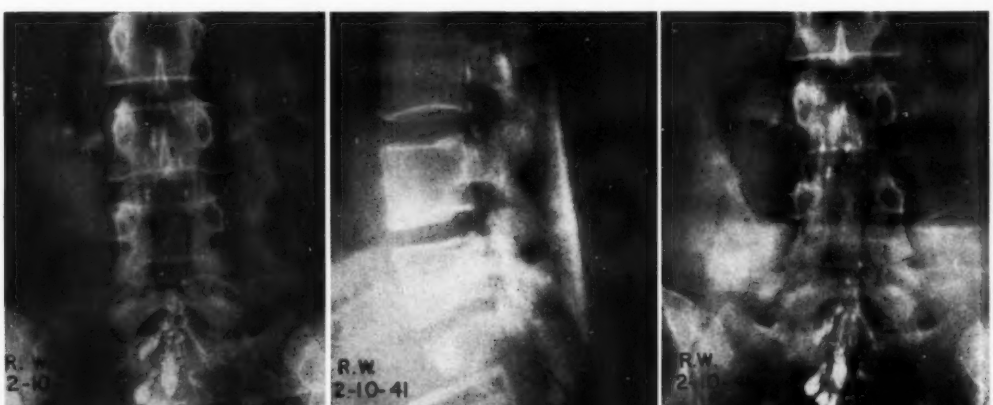


Fig. 4. R. W. Repair of old laminectomy defect in 4th lumbar by double clothespin graft from the 3d lumbar to the 5th lumbar.

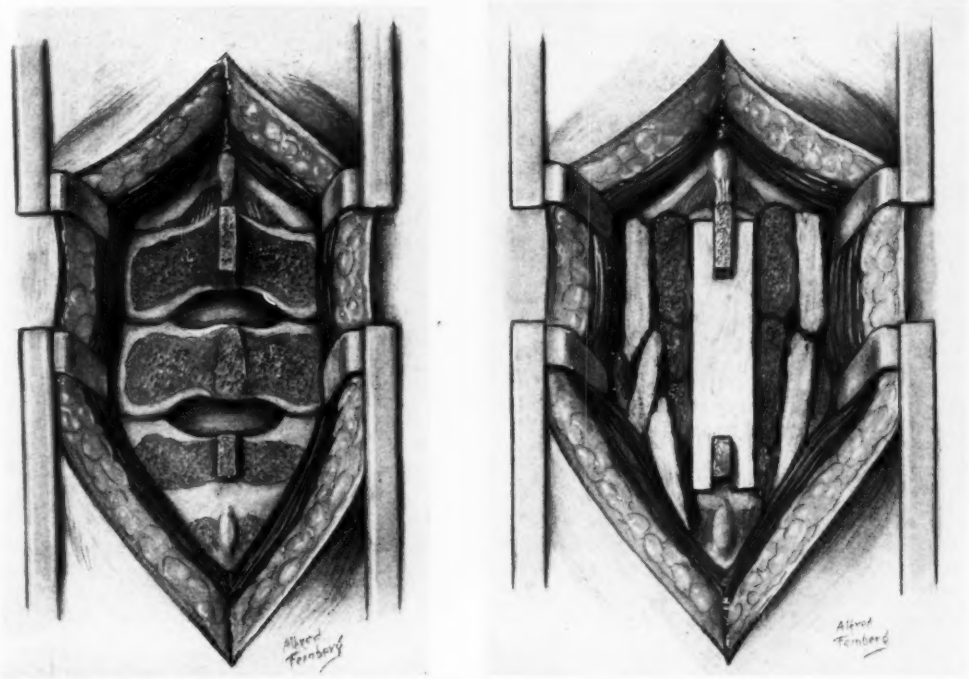


Fig. 5. a, left, Shows the preparation of the posterior elements of spine with squaring off of spinous processes of the 4th lumbar and the 1st sacral and the removal of the spinous process of the 5th lumbar. b, The implantation of a double clothespin graft which is reinforced with iliac chips.

laminal arches left behind. The patient should be placed upon a table in a flexed position, so as to spread the spinous processes apart. The spinous processes should be squared off on their lateral surfaces and on the margin which will be in contact with the graft. The opposing margins of the spinous processes between which the graft is to be laid should be slightly undercut. When the patient is in the extended position, the graft will then be forced to a deeper position rather than be extruded by pressure. After the spine preparation has been carried out, a malleable probe can be bent accurately to fit between the spinous processes which are to be included. A clothespin graft can be cut from the upper tibia, the abutting portion of the graft being cut accurately to the length of the previously bent probe or measure. The abutting or slotted ends of the clothespin graft should be cut at a slight bevel to match the undercut margins of the spinous processes. This will aid in retention of the graft *in situ* when the patient is straightened out. When the graft extends below the 5th lumbar segment, the deep borders of the distal clothespin prongs will have to be beveled up sharply to match the dorsal curve of the sacrum. Occasionally, to secure a good strong base on the sacrum, it is necessary to by-pass the 1st sacral and anchor the graft to the 2d sacral spinous process. When previous fusions have been done, the upper border of the previous fusion may be undercut and notched transversely. The tibial graft is cut as a single clothespin above with a beveled lower border (Fig. 1, d). The graft can then be sprung between the notch in the previous fusion below and the spinous process above. Reinforcement of the clothespin graft with iliac bone is desirable always in spondylolisthesis and generally when used in association with laminal defects. If laminal defects are

present, it is necessary to set the clothespin graft as tightly down on the base of the spinous process as possible, so that iliac or other chip grafts laid in along the side will not fall in the laminectomy wound and become a potential source of extradural pressure. Following its implantation an accurately fitted clothespin graft can be immediately tested for stability by grasping and shaking it with a bone holding forceps. The amount of stability secured without any other fixation is rather startling. Routine closure of the wound is carried out and a jacket used if desired.

Any spinal operation is apt to be a massive procedure, and this is no exception. Infusions and transfusions if necessary should be done.

CONCLUSIONS

1. A graft is proposed which by its shape and position is maintained in the spine tightly fixed in place without using extraneous material.

2. By the position and the normal mechanics of the lumbar spine, distraction is secured not only of the posterior elements of the spine but of the anterior elements as well.

NOTE.—Since the submission of this article I have received a personal communication from Dr. Alexander Gibson of Winnipeg, Canada, calling attention to an article entitled "A Modified Technique for Spinal Fusion," written by him and published in the September, 1931 issue, vol. 53, pp. 365-369. This article clearly entitles Dr. Gibson to priority; we do not claim originality for the graft as presented, but do believe that its use should be restricted to the repair of laminal defects and for spondylolisthesis.

RELATIONSHIP BETWEEN THE CLINICAL AND ROENTGENOLOGICAL FINDINGS IN BONE METASTASES

J. BORAK, M.D., New York, New York

IN their monograph, *The Vertebrae*, George and Leonard state: "Repeated examinations lasting over a fairly long period of time may be made without finding any visible changes in the body of the vertebra that is clinically suspected."

The usual history is that, following mastectomy to remove a malignant tumor, after varying intervals of time the patient begins to suffer from pains along the sciatic nerves which are not relieved by the usual methods of treatment. A metastatic lesion is then suspected and an x-ray examination is made. Although the examination is carried out in accordance with all the rules of roentgenological technique, the result may be negative. In 1 case under my observation, a 48 year old woman, who had been operated upon for a breast cancer about a year ago, died of pneumonia 3 weeks after a roentgenological examination of the spine had revealed no metastasis. The postmortem x-ray examination of the excised vertebra was likewise negative. Even the x-ray examination of thin sections of the dried vertebrae disclosed no metastatic lesion (Fig. 1). Yet, on microscopical examination cancer tissue was found in abundance (Fig. 2). Thus the following triad presents itself: clinically, there is pain; microscopically, cancer; and roentgenologically, a normal appearance.

Erdheim was so impressed by the discrepancy between the roentgenographic and microscopic pictures that he developed the following theory: He assumed that, besides the osteolytic and osteosclerotic types of metastasis of bone, there is a third type of metastasis not influencing the bone in a destructive or a productive sense. Erdheim called these metastases osteoindifferent. Konrad Weiss, supporting Erdheim's theory, called them osteoneutral.

From the Department of Radiology, New York University Medical College.

However, the following two objections may be raised against the theory of Erdheim: (1) There are cases in which the roentgenological findings in the spine are negative but in which roentgenological findings in other bones, for instance in the skull, are positive. (2) There are cases in which the findings at the first roentgenological examination are negative, while in subsequent examinations the findings are positive.

In view of these facts, it is obviously not permissible to assume that there exists a special type of cancer tissue, supposedly indifferent to bone. Rather we should assume that there is a roentgen-negative stage in bone metastasis, comparable to the Wassermann-negative stage in syphilis. Sicard, Coste, and Belot use the term "preradiologique phase."

The occurrence of a roentgen-negative stage in the development of bone metastatis may be explained as follows: As is well known, tumor cells, brought to the bone by the blood stream via the nutrient arteries, settle primarily in the wide spaces of the spongy portion, thus the dense, compact portion of bone is affected primarily only in rare instances.

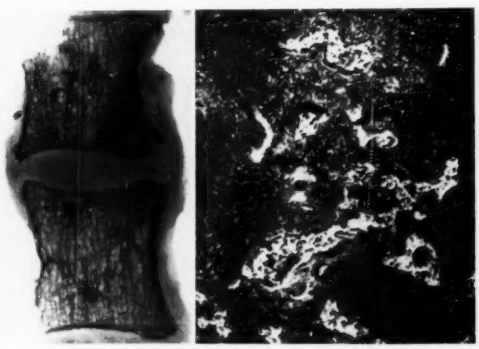


Fig. 1. Left, Roentgenogram of thin sections of vertebra showed no metastatic lesion.

Fig. 2. Photomicrograph of section of same vertebra as shown in Figure 1 shows abundant cancer tissue.

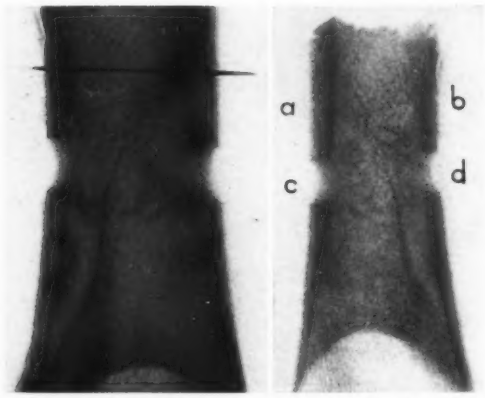


Fig. 3. Left, Tubular bone punctured by needle.
Fig. 4. Same bone as in Figure 3. *a-b*, Canal produced by needle distinct in compact bone but no trace is visible in spongy central portion. *c-d*, Canal made by 1 centimeter drill plainly evident in compact bone but not visible in spongy bone.

This is probably the reason why the vertebrae, being particularly rich in spongy bone, are the site of predilection of metastases. It may be added that contrary to the description in many textbooks "A proper, firmly coherent layer of compact bone, comparable to that of long, tubular bones, does not exist in the body of the vertebra" (Junghans).

Accordingly, roentgenograms of the spine, particularly lateral views, clearly show that the vertebral body consists exclusively of spongy bone. The dense, ring-like shadows on the upper and lower surfaces of the vertebra belong genetically to the intervertebral disc, and fuse but secondarily with the vertebra, as Schmorl has shown. These shadows are less dense in persons under 15 and in the aged.

The fact that the vertebra consists exclusively of spongy bone has a decisive bearing on the roentgenological visualization of lesions occurring within them, for the reason that, although both the spongy and the compact portions consists of calcium-containing bone tissue, there is an important difference between the structures of the two parts. The spongy bone forms a wide meshwork of very thin trabeculae, while the compact bone forms a thick layer of dense bone tissue. Consequently, the spongy bone produces a shadow of much less density than the compact bone.

The following experiment illustrates the roentgenological consequences of this fact:

Half of a dried tubular bone consisting of both a well developed compacta and spongiosa was punctured with a needle (Fig. 3). The roentgenogram shows that the canal produced by the needle is quite distinctly seen in the compact portion of the bone, while no trace of the canal is visible in the spongy portion (Fig. 4 *a-b*). In another experiment the same result was obtained when the canal was pierced by a drill 1 centimeter in diameter (Figs. 3 and 4, *c-d*). These experiments prove that a defect which is visible in a compact bone may be quite invisible in a spongy bone.

In the light of these experiments it becomes clear that lesions in the vertebrae may go undetected since, as stated, the latter consist exclusively of spongy bone. An experiment performed on a dried vertebra shows that even as many as 5 defects of varying length and width extending in craniocaudal direction (Fig. 5) remain invisible on films taken in anteroposterior, lateral, and oblique positions (Figs. 6, 7, 8), representing all the directions in which vertebra can be examined roentgenologically *in vivo*. Similar observations have been published by Chasin in Russia; Boehmig and Previg, in Germany; Snure and Maner, here.

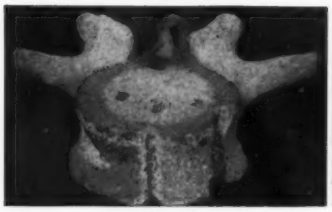


Fig. 5.

Fig. 6.

Fig. 7.

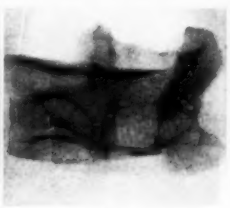


Fig. 8.

Fig. 5. Illustration of dried vertebra in which 5 defects are visible.

Figs. 6, 7, and 8. Roentgenograms of vertebra in Figure 5 taken in anteroposterior, lateral, and oblique positions.

The question immediately arises, nevertheless, why certain pathological conditions of the vertebra are well recognizable on x-ray films. Our experiments on dried vertebrae and observations on clinical cases have led to the following conclusion: A defect in the vertebral spine can be seen roentgenologically because of 2 things: (1) The defect is portrayed orthoroentgenographically, that is, the rays traversing the defect are parallel to its longest axis; (2) the defect changes the contour of the vertebra.

A closer analysis of the first condition reveals the following results:

1. A defect produced artificially in a dried vertebra which extends through less than one-third of the vertebra cannot be demonstrated roentgenologically. If the defect exceeds one-third but does not extend through the whole vertebra, it appears merely as an area of decreased density (decalcification or rarefaction), because the remaining bone tissue still casts a shadow (Fig. 9, *a*, *b*). To be demonstrated as such, a defect must extend throughout the whole bone in the direction of the rays (Fig. 9, *c*, *d*, *e*).

2. Besides the extent of the defect, a thick-walled abdomen also has an important bearing on roentgenological visibility. In the case of a thick abdominal wall the statement of Ferguson applies; "in some spine lesions decalcification may not be recognizable because of the great thickness through which the spine is viewed." My experience has shown that if, in a given direction, the thickness of the tissues overlying the vertebrae exceeds 20 centimeters, only a defect affecting half the vertebra in the direction of the central ray will make its appearance roentgenologically as an area of lessened density. Lesions not discernible clinically do, however, become obvious on postmortem examination of the excised vertebra (Fig. 10).

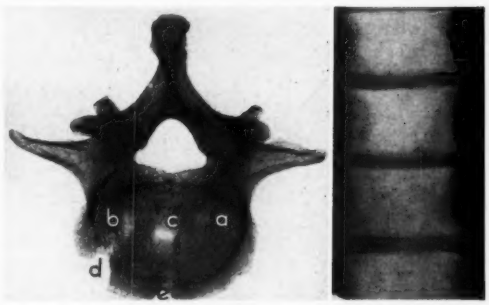


Fig. 9. Left, *a*, *b*, Defects extending through one-third of vertebra appear as areas of decreased density; *c*, *d*, *e*, defects extending through whole bone.

Fig. 10. Postmortem examination of excised vertebrae.

A closer analysis of the second condition—that the defect is visible because it changes the contours of a vertebra (Fig. 11)—reveals the following:

1. A defect which changes the contours of a vertebra can be distinguished as such roentgenologically regardless of its extent. In its initial stages the contour, usually smooth, appears blurred (Fig. 12), later the contour, normally archlike, appears angulated (Fig. 13). In an advanced stage the diameter of the vertebra also is reduced so that the size is changed too (Fig. 14). Then not only the

Fig. 11. Fig. 12. Fig. 13. Fig. 14.

Fig. 11. Contour of vertebra changed so that defect is plainly visible.

Fig. 12. Initial stage, blurred contour.

Fig. 13. Later stage, angulated contour.

Fig. 14. Advanced stage, diameter of vertebra is here reduced.

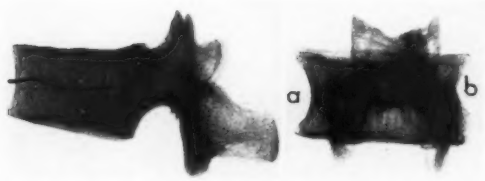


Fig. 15. Left, Defect in dried vertebra marked by wire.
Fig. 16. *a*, Lesion on left side; *b*, lesion on right side.

contour but the shape of the whole vertebra is altered. The most frequent deformity resulting from the collapse of the vertebra is the wedge form. A reduction of the size of the vertebra without change of its shape is very rare.

2. The thickness of the overlying abdominal tissues does not interfere with the roentgenological manifestation of a contour-changing defect. If the vertebra is presented distinctly, even the smallest contour-changing defect can be recognized roentgenologically.

The subsequent experiment illustrates the difference between the appearance of a defect of the vertebra visible because it is portrayed orthoroentgenographically and that of a defect visible because it had changed the contours of the vertebra. In a dried vertebra a defect—marked by the wire—was produced on the left side, extending through its whole length in the anteroposterior direction (Fig. 15). On the right side of the vertebra a small lesion was produced, transforming the normally archlike contour into an angulated one. Roentgenograms show that the lesion on the left side (Fig. 16, *a*), despite its extent, can hardly be seen while the small defect on the right side is distinctly visible (Fig. 16, *b*). In addition the lesion on the left side, though extensive, would in all probability be completely undetected if the vertebra were examined through the abdomen, whereas the defect on the right side, despite its smallness, would remain visible roentgenologically as long as the vertebra is visible.

The conclusion to be drawn from these observations is that only in a very advanced stage can a lesion in the vertebra become visible due to its extent. On the other hand, even the smallest defect can be recognized roentgenologically if it causes a change in the contour of the vertebra. In other words, a defect seen because it is portrayed orthoroentgenographically is a *late* x-ray finding; a defect seen because it changed the contours of the vertebra is an *early* x-ray finding.

Hence the practical rule: In searching for an early sign of a spinal metastasis we should not expect to find decalcification or deformity. Let us first of all watch the contours of the vertebrae. If there is evidence of the slightest change, such, for instance, as a blurred or angulated contour, diagnosis of a spinal metastasis may be regarded as assured.

What has been said of the osteolytic metastases in the vertebra applies equally to

Fig. 17. Roentgenogram of pelvis—no pathological change evident.

Fig. 18. Same patient as in Figure 17 a few weeks later showing definite change.

Figs. 19, left, and 20 Pathological fracture occurred in spite of fact that roentgenogram taken a short time before revealed no metastasis.

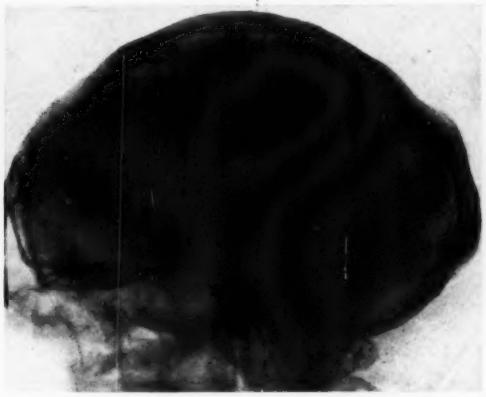


Fig. 21. Roentgenogram showing extension of metastases to skull.

osteolytic metastases in other bones with a similar structure, namely, a well developed spongy bone with lacking or only poorly developed compacta. In this category are the ribs, the pelvis, and the metaphyseal parts of the long bones. A roentgen-negative stage exists in the development of a metastasis in these regions, during which severe pain may be present while the x-ray films do not reveal any pathological changes (Fig. 17). A few weeks later, however, the x-ray film may become positive (Fig. 18). In the long bones such as the femur or humerus even a pathological fracture may occur, though the x-ray films, taken a short time before, did not give the slightest indication of the presence of metastasis (Figs. 19, 20).

The contrary holds true of bones with a structure opposite to that of the vertebra, that is, bones with a well developed compacta and comparatively less developed spongiosa. Here belong such parts of the skeleton as the skull and the diaphyses of the long bones. No roentgen-negative stage does exist in the development of a metastasis in these regions. Whenever clinical symptoms pointing to a metastasis are present, roentgenological signs are also encountered. What is more, metastases are roentgenologically visible in these regions even before any clinical symptoms are present. They are frequently encountered only incidentally, for instance, when one has detected spinal metastases and subsequently is searching for metastases in other bones. One is surprised time and again about the extension of the metastases, for instance, in the skull (Fig. 21), while no pain and no other symptoms whatsoever are present.

The same holds true of the osteosclerotic metastases, regardless of their site. Osteosclerotic metastases, found with particular frequency in cancer of the prostate, may be

Fig. 22. Metastases following cancer of prostate.

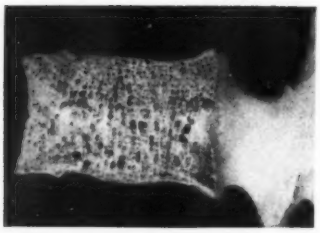


Fig. 23.

roentgenologically well pronounced while clinically no symptoms are present at that time (Fig. 22). The spinal metastases are no exception to this rule. One may assume that in a spongy bone such as the vertebra an osteosclerotic metastasis produces a contrast of density to its surroundings, as it normally exists in other bones between the compacta and spongiosa (Fig. 23). Therefore, it is a wise procedure in cancer of the prostate to take films of the vertebrae before an operation is performed, because metastases may be present in absence of any clinical symptoms.

CONCLUSIONS

In conclusion we may say that if we examine the relationship between the clinical and roentgenological findings in bone metastases, two groups can be distinguished:

One group comprises metastases which give clinical symptoms but are invisible roentgenologically. These may be called *roentgenologically occult* metastases. The other group of metastases may be roentgenologically visible but not cause clinical symptoms. These may be called *clinically silent* metastases.

It appears that whether a metastasis belongs to the one or the other group depends neither on the nature of the primary tumor nor upon the constitution of the patient but exclusively upon the structure of the affected bone, that is, on the relationship between the amount of the compact and spongy bone tissue.

REFERENCES

1. Boehmig and Previg. Fortsch. Röntgenstrahl., 1931, 43: 541–579.
2. Chasin. Fortsch. Röntgenstrahl., 1928, 37: 529–535.
3. Erdheim. Virchows Arch., 1929, 275: 383–396.
4. Ferguson. Roentgen Diagnosis of the Extremities and Spine. Annals of Roentgenology, New York: Paul B. Hoeber, Inc., 1941.
5. George and Leonard. The Vertebrae Roentgenologically Considered. Annals of Roentgenology. New York: Paul B. Hoeber, Inc., 1929.
6. Junghans. Pathologie der Wirbelsäule in Handbuch d. path. Anat. und Histol. IX/4, 1939, 216–411.
7. Schmorl and Junghans. Fortsch. Röntgenstrahl., 1932, Ergaenzungsband 42.
8. Snure and Maner. Radiology, 1937, 28: 172–177.
9. Sicard, Coste, and Belot. Paris méd., 1931, 79: 550–556.
10. Weiss. Fortsch. Röntgenstrahl., 1933, vol. 48.

PROLAPSE OF THE EFFERENT (DISTAL) SEGMENT OF BOWEL AFTER COLOSTOMY

JOHN L. KEELEY, M.D., F.A.C.S., Chicago, Illinois

THE purpose of this report is to present the case histories of 5 patients in whom prolapse of the efferent (distal) segment of bowel occurred following colostomy and to discuss the etiology, pathogenesis, and treatment of this complication.

The literature contains numerous references to instances of prolapse of the afferent or proximal segment of bowel following colostomy, but reports describing this complication distal to the colostomy are decidedly infrequent. Bevis, in reporting his case of prolapse involving the distal segment, stated that he had found only 3 reports of this complication in the literature and all were in French journals (4, 10, 11). Antonioli reported 1 case in 1935. Gabriel and Lloyd-Davies, in reviewing the complications in 500 patients for whom palliative colostomy was performed because of inoperable carcinoma, reported 12 instances (2.4 per cent) of prolapse of all types. They considered it a rare complication and stated that it usually takes place from the upper opening, occasionally from the lower opening, and sometimes from both. Veal in 1940 reported 4 instances of prolapse of the sigmoid colon following colostomy. The lower segment was involved in 3 of the 4 patients.

The following case reports and illustrations present the outstanding aspects of this type of prolapse.

CASE 1. C. G., a 44 year old unemployed colored painter, was first admitted to the Louisiana State University surgical service in the Charity Hospital at New Orleans on March 3, 1939. At that time a tumor mass which almost completely occluded the rectal lumen was found, and the diagnosis of carcinoma of the rectum was verified by examination of a biopsy specimen. On March 22, 1939, under spinal anesthesia, the abdomen was opened and exploration disclosed widespread peritoneal seeding. A simple loop colostomy, in which the sigmoid colon was used, was made through a left McBurney incision. The wound healed without infection. The colostomy was opened on the 15th day after operation; it functioned well and the patient was discharged on April 11, 1939.

On May 1, 1939, he was readmitted for radiation therapy. His condition was essentially the same as on the previous admission. On the morning of the 3d day in the hospital, while the patient was getting out of bed, the distal loop of the colostomy prolapsed. The picture shown in Figure 1 was taken within the next 2 hours. The prolapse was reduced in the usual manner (Fig. 2). There was no recurrence although the stenosis in the rectum persisted. Ascites and general weakness developed and the patient died at his home on June 14, 1940, about 15 months after first admission. No autopsy was done.

CASE 2. E. D., a 30 year old colored female cook, was first admitted to the Louisiana State University surgical service of the Charity Hospital of New Orleans on August 19, 1940, with the chief complaint of constipation of 2 years' duration. She was found to have a firm annular stricture of the rectum, and a diagnosis of rectal stricture due to lymphopathia venereum was made. On August 24, 1940, a simple loop colostomy was made, the sigmoid colon being used. It was opened 3 days later and functioned well from the beginning. The patient was discharged on September 9, 1940.

The patient was next admitted on October 21, 1941, because of prolapse of the distal loop of the colostomy. The protruding segment of bowel was then about 3 inches (7.5 cm.) long. Because of a strongly positive Wassermann reaction, repair of the prolapse was deferred. The bowel was undisturbed and the patient was discharged with provisions for a course of antiluetic therapy.

When the patient was readmitted on February 3, 1941, the prolapse had increased in size (Fig. 3) and, according to the history, had been almost 8 inches (20 cm.) long at times. The prolapse was reduced. Because of extensive rectal and perirectal involvement, an abdominoperineal resection of the rectum was done, a stump of sigmoid being left distal to the colostomy. This stump was turned inside out and protruded from the colostomy stoma in the same manner as had the prolapse. Five days later the protruding portion of bowel was freed from the abdominal wall, the layers of which were then closed snugly about the proximal loop making an end type of colostomy. The excess bowel was removed with cautery. The end-result is shown in Figure 4.

From the Department of Surgery, Louisiana State University School of Medicine, and the Louisiana State University Surgical Service, Charity Hospital, New Orleans, Louisiana.

Fig. 1. Case 1. Prolapse of the distal segment of a left inguinal loop colostomy. The opening into the proximal segment is plainly seen. The prolapsed portion is swollen and edematous.

Fig. 2. Reduction of the prolapse is accomplished in this manner. The maneuver is essentially the same as that employed in reducing a rectal prolapse with the exception that, due to its length, the prolapsed bowel must be supported. The use of dry gauze as illustrated facilitates the reduction.

Case 3. V. B., an unemployed 19 year old colored girl, was admitted to the Louisiana State University surgical service in Charity Hospital at New Orleans on February 26, 1941, because of almost complete lower bowel obstruction. An annular stricture was found about 2 centimeters above the anal orifice. At this point the lumen of the bowel admitted nothing larger than a No. 12 urethral catheter. The Frei test was positive. A diagnosis of rectal stricture due to lymphopathia venereum was made. Because of the obstruction, it was necessary to make a colostomy, the sigmoid colon being the site selected. This was done on March 5, 1941, under general anesthesia. The colostomy was opened 8 days later, the wound having healed without infection. One week later the patient was discharged from the hospital.

The patient was readmitted on April 6, 1941, complaining that the bowel had "fallen out and was painful." The distal loop of the colostomy was found to be prolapsed, as shown in Figure 5. The prolapse was about 15 centimeters long, engorged, and edematous. It was reduced and within the next 2 days there were 4 small bowel movements through the rectum. A flat gauze dressing was applied over the colostomy stoma and held in place by a wide support of adhesive tape. The patient was allowed to be up and was discharged a few days later.

On May 19, 1941, the colostomy stoma was almost completely closed (Fig. 6). The bowels were moving through the rectum although the stricture was essentially unchanged. Similar findings were present on August 21, 1941.

Case 4. R. S., a colored female, age 34 years, was admitted to the Louisiana State University surgical service of Charity Hospital at New Orleans on April 18, 1941, with the chief complaint of constipation, rectal pain, and passage of blood and pus per rectum. Examination revealed a complete annular stricture of the rectum about 6 centimeters from the anal orifice. The lumen at this point was less than 1 centimeter in diameter. A diagnosis of rectal stricture due to lymphopathia venereum was made and on April 29, 1941, the first stage of a modified Lahey procedure was done, the distal stump of bowel being implanted in the lower end of the left rectus incision. An uneventful recovery followed and the patient was discharged on May 20, 1941.

On the second admission, July 1, 1941, the patient complained of recurrent protrusion of the lower segment. The exposed mucosa was very edematous and bled easily when manipulated. The prolapse was reduced manually (Fig. 2) and remained reduced under a regimen of restricted activities while on the ward. On July 16, 1941 the second stage of the resection was done. The patient was discharged on August 7, 1941. She was seen on August 27, 1941, and reported a gain of 5 pounds and considerable increase in strength and appetite. The colostomy functioned satisfactorily, and the anterior and posterior wounds of the abdominoperineal resection were solidly healed.

Case 5. L. W., a colored female, age 50 years, was admitted to the Louisiana State University surgical service of the Charity Hospital at New Orleans on March 4, 1940, complaining of rectal pain, constipation, and sanguinopurulent discharge from the anus. No stricture or mass was palpable on digital rectal examination, but with the proctoscope an annular stricture, through which blood and pus drained, was found 10 to 11 centimeters above the anal orifice. Moderate hypochromic anemia was present. The Frei test was reported to be negative. A barium enema disclosed an irregular filling defect compatible with a diagnosis of carcinoma, but

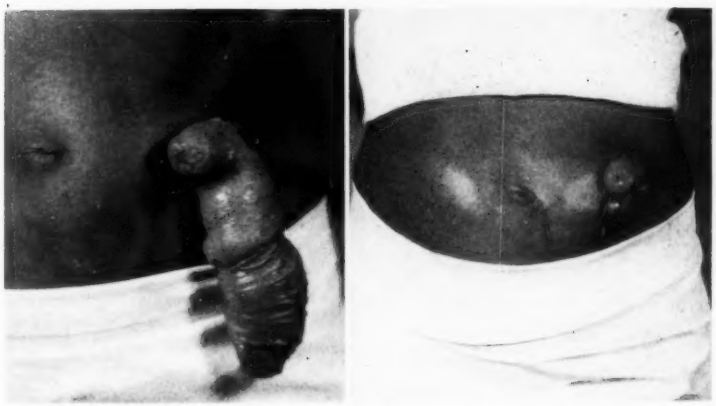


Fig. 3. Left, Case 2. Prolapse of the distal segment after simple loop colostomy. The proximal loop shows a mild degree of prolapse also.
Fig. 4. Case 2. The end-result following abdominoperineal resection of the rectum and plastic repair of the colostomy.

biopsy specimens from the area failed to substantiate this impression. On March 15, 1940, a laparotomy was performed. The findings suggested that the mass at the rectosigmoid junction was due to inflammation rather than neoplasia. The adjacent mesosigmoid was shortened and thickened. A modification of the Lahey procedure was done. Recovery was uneventful, and the patient was discharged on March 23, 1940.

The patient was next admitted on August 23, 1940, complaining of prolapse of the distal loop of the colostomy. During the interval, the colostomy had functioned satisfactorily. Considerable pain had been present, however, since the onset of the prolapse. The blood picture had definitely improved. On September 6, 1940, under general anesthesia, the stump of the distal loop was freed from the abdominal wall and the peritoneal cavity was entered. The mass at the rectosigmoid junction had decreased in size and the subsidence of inflammation in the mesosigmoid had left that structure thinner and longer resulting in increased motility of the sigmoid. The distal loop was drawn taut and the abdominal wall was closed snugly about it. After healing had taken place the protruding portion of the bowel was removed with the cautery. Although a mild mucosal prolapse persists the patient has been free from discomfort and pain.

ETIOLOGY AND PATHOGENESIS

The etiological factors in prolapse of the distal segment of bowel after colostomy may be arranged as follows:

1. Excessively large defect in the abdominal wall due to: (a) failure to close the abdominal wall snugly about the protruding loop of bowel; (b) loss of tissue from the edge of the defect in the abdominal wall because of infection and sloughing; (c) relaxation of the abdominal wall in patients with atrophic or atonic tissues.

2. Redundant bowel due to: (a) Lengthening of lower segment because upper segment usually is drawn so taut that it cannot prolapse; (b) resolution of inflammation in either shortened bowel or mesosigmoid below the colostomy site which may be followed by increase in length and motility of the distal segment.

3. Obstruction of lower segment.

4. Increased intra-abdominal pressure.

It is obvious that a combination of factors is concerned in the development of prolapse. However, the factor which has been considered to be most important by some authors is the size of the opening in the abdominal wall. This appears to be especially significant in the Maydl-Reclus type of loop colostomy in which the two limbs of bowel pass through a common defect. Most of the reported instances of prolapse (1, 2, 4, 10, 11, 14) and 3 of the 5 cases described in this report have been complications of this type of colostomy. Weakness of the abdominal wall is commonly seen as a part of general weakness and debility in patients with advanced carcinoma of the rectum or long standing lymphopathia ven-

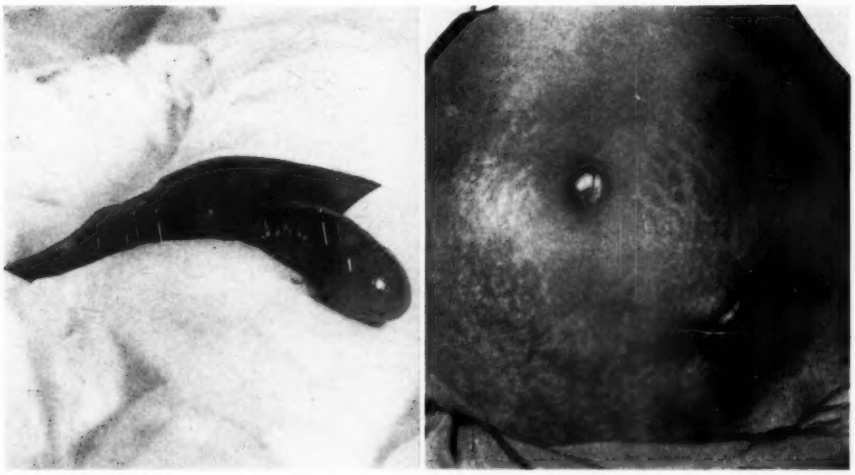


Fig. 5. Left, Case 3. Prolapse of the distal segment after loop colostomy for high grade obstruction caused by rectal stricture.

Fig. 6. Case 3. After reduction of the prolapse and a period of bed rest contraction about the colostomy wound led to its almost complete closure within 6 weeks.

ereum with rectal stricture and secondary infection. The size of the defect may permit a mild degree of prolapse initially, but the stretching effect of the protruding bowel often enlarges the defect so that prolapse recurs or persists. The opening in the abdominal wall may become too large due to loss of substance from the edge as a result of infection and sloughing. Unfortunately this is likely to happen in the group of patients who already have relaxed or thin abdominal walls or both so that a gaping defect is the end-result.

Redundancy of the distal segment of bowel and mesosigmoid is a necessary, but not the sole, factor in the development of prolapse. The common practice of rendering the proximal segment taut to prevent prolapse of that part of the bowel when making a simple loop colostomy of the Maydl-Reclus type leaves the distal portion long enough to prolapse. The degree to which such a segment will prolapse depends to some extent on the length of the mesosigmoid. This point is well illustrated by Case 5 in which an inflammatory lesion obstructed the rectum and involved the mesosigmoid, shortening and thickening the latter. Prolapse did not occur until 5 months after the colostomy had been made. When the prolapse was repaired the inflammatory process was found to have subsided considerably, and thinning and elongation of the mesosigmoid had occurred. Along similar lines Loubat and Fontain describe a patient with complete stenosis of the rectum due to inoperable carcinoma. In performing colostomy it was barely possible to bring the sigmoid through the abdominal wound. No difficulty was encountered from prolapse in this instance, although the stenosis remained complete.

Obstruction of the lower segment (usually in the rectum) was a prominent feature of many of the reported cases (4, 10, 11, 14). In all of the cases described in this report the rectal lesion, whether carcinoma (1 case) or rectal stricture due to lymphopathia venereum (4 cases), caused almost complete obstruction of the rectum.

The rôle of increased intra-abdominal pressure is difficult to evaluate as a single factor, but in combination with a gaping defect in the abdominal wall, prolapse may be initiated in much the same way as the bulging in certain forms of hernia.

In the absence of a gaping defect in the abdominal wall, the actual mechanism of pro-

lapse of the distal segment of bowel is similar to that of intussusception as Veal points out. Discharges from an ulcerated lesion or quantities of mucus accumulate in the rectum and because of obstruction reversed peristalsis occurs. This everts the redundant portion of the bowel through the fixed part (at the colostomy site). The prolapsed bowel, which represents the intussusceptum, escapes through the colostomy stoma instead of being enclosed by the intussuscipiens as occurs in the usual process of intussusception. In prolapse, therefore, there is no intussuscipiens. For the sake of clarity in describing the operative correction later on, the prolapsed portion of bowel may be called the intussusceptum and therefore quite properly can be said to have an inner and an outer layer.

The fully developed prolapse measures from 15 to 20 centimeters in length. The diameter varies from 3 to 7 or 8 centimeters depending upon the degree of interference with the circulation. Within a few hours after the prolapse the bowel becomes edematous, the mucosa is shiny and is covered with mucus. As the return of venous blood is interfered with, the bowel becomes so engorged that it bleeds readily when manipulated. If allowed to remain unreduced the prolapse may become irreducible and circulatory changes leading to ulceration and gangrene may occur. In some instances the size of the prolapse is such that it obstructs the proximal segment completely and manifestations of intestinal obstruction may appear. There is usually some pain or discomfort associated with prolapse, but as a rule it is not severe.

TREATMENT

The most important prophylactic measures concern efforts to control the size of the defect in the abdominal wall. Toward this end, particular attention should be directed to fitting the edges of the incision snugly about the loop of bowel. In these patients who so often are in poor general condition, the careful observance of the points in wound healing as emphasized by Whipple and Reid (12) is especially important in securing primary healing. Thus, if time permits, any existing anemia, hypoproteinemia or vitamin deficiency, particularly lack of vitamin C, should be corrected prior to operation.

Every effort should be made to avoid infection as it may be followed by sloughing which results in an unnecessarily large opening. The possibility of infection may be decreased by minimal manipulation of the bowel and by completely avoiding the use of sutures in any layer of the bowel wall, fibrinous exudate being allowed to attach the bowel to the wound edges.

The substitution of a flexible rubber tube for the glass rod so commonly used in preventing the retraction of the loop of bowel in the Maydl-Reclus type of colostomy may prevent the development of an area of pressure necrosis in the bowel at the mesenteric border should abdominal distention develop. Leaking through such an area of necrosis often allows concealed contamination of the wound and serious infection may develop.

Since rendering the proximal segment of the sigmoid taut in making a Maydl-Reclus type of colostomy has been so effective in preventing prolapse of the afferent loop, the possibility of similarly caring for the distal segment has been considered. This would necessitate the exteriorization of a relatively long segment of bowel which would ultimately require removal, a sort of Mikulicz procedure. As Loubat and Fontain pointed out, the low incidence of a relatively benign complication (prolapse) does not warrant substituting this more formidable procedure for the simple one of constructing a loop colostomy.

Following the construction of the colostomy causes of increased intra-abdominal pressure, such as chronic cough, straining at urination or defecation, lifting heavy objects, or other similar exertion, must be avoided or kept at a minimum. A colostomy in reality represents a defect in the abdominal wall which is plugged by a loop of bowel. Even when the "plug" fits snugly into the defect the site of the colostomy is a weak area which does not withstand intra-abdominal pressure as well as the intact abdominal wall. Under the influence of increased intra-abdominal pressure the loop of bowel may dilate the defect in the abdominal wall in a manner similar to the omental wedge which is frequently an im-

portant factor in the development of hernia.

The prolapse may be treated palliatively by reduction as shown in Figure 2. The patient is given appropriate doses of morphine to aid in relaxation of the abdominal musculature after which reduction is usually not difficult. Elevation of a markedly swollen prolapsed bowel may be followed by some decrease in size. Rather firm pressure if applied uniformly to the apex of the prolapse may so decrease its size that reduction may be initiated, and, once started, no difficulty is usually encountered in completing it. After reduction the patient should be kept in bed for several days and when allowed to be up should wear a flat piece of gauze instead of a colostomy bag over the colostomy stoma. In addition, a wide abdominal support should be worn. Early reduction as in Cases 1 and 3 will prevent stretching the defect in the abdominal wall and thus guard against recurrences.

The radical treatment of prolapse varies considerably. Plastic procedures designed to correct a gaping defect in the abdominal wall may be successful occasionally but often fail because of wound infection or because of poor wound healing in an area in which previous infection has resulted in considerable scar tissue. However, should this type of repair be followed by satisfactory healing, the redundant distal loop and stenosis which remain uncorrected make recurrences very probable.

Amputation of the prolapse is the most frequently practiced method of treatment. The procedure employed is exactly like that advocated by Mikulicz for the correction of complete prolapse of the rectum (3), and consists of amputating the intussusceptum near the surface of the skin, only enough cuff of outer layer being left to suture to the open end of the inner layer. As the division of the inner layer proceeds, the resulting proximal free edge is sutured to the cuff of outer layer to prevent retraction. The completion of this row of sutures which has approximated the serosae of the inner and outer layers closes the peritoneal cavity. By division of the outer layer of the intussusceptum near the skin, an adequate blood supply to the remaining narrow cuff of bowel is assured and, at the completion of the operation, the suture line is held at the level of the skin. A longer outer cuff may have poor blood supply and, should the site of anastomosis finally rest inside the peritoneal cavity, a leak from the suture line may lead to generalized peritonitis. This series of events led to the death of Charbonnel's patient, whereas the usual type of repair with the suture line at the level of the skin was entirely satisfactory in cases reported by Bevis, Loubat and Fontain, and Madranges.

The procedure of ligating the intussusceptum about a hard rubber tube placed in its lumen has been applied to complete prolapse of the rectum (13) and to prolapse of the afferent segment of bowel in a loop colostomy by Hall. Although rather severe cramp-like abdominal pain occurred after the ligatures were applied in Hall's case, the treatment was successful, the intussusceptum coming away 5 days later and complete healing followed without infection or hemorrhage just as in the cases of complete prolapse of the rectum reported by Reid (13).

Veal reports an instance of fatal peritonitis following cautery excision of what was thought to be a prolapse of mucosa at the site of a loop colostomy. Because of this experience, Veal has proposed a two stage operation. The first stage is carried out through a left rectus incision opposite the colostomy site and consists of attaching the serosa of the outer layer of the intussusceptum to the serosa of the inner layer by means of a series of interrupted sutures placed at the level of the parietal peritoneum. This seals off the peritoneal cavity from the potential space between the inner and outer layers of the intussusceptum and after several days the second stage of the operation, which is essentially the Mikulicz procedure for complete rectal prolapse, is done.

The plan of radical treatment will of course depend on the type of colostomy. If prolapse occurs in the distal segment after a defunctionalizing colostomy of the Lahey or Devine type, the prolapse may be attacked directly as it has already been protected from gross and continuous contamination and postoperatively will not be subjected to this hazard. The prolapse in the fifth patient was managed in this way.

Undoubtedly many fatalities following surgical treatment of the prolapsed distal segment of a loop colostomy have remained unrecorded as only Veal's and Charbonnel's cases were found in the literature. From the experiences of Bevis, Loubat and Fontain, and Madranges the direct attack on the prolapsed segment appears to be satisfactory. In the future it is possible that the risk of peritonitis in these cases may be lessened by the use of chemotherapeutic agents (6).

Although 2 of the cases of prolapse in this series were treated by abdominoperineal resection it should be emphasized that such radical procedure is not advocated for prolapse *per se*. In the cases thus treated the primary object was to remove a focus of infection for which all previous treatment had been unsuccessful. The correction of the prolapse was of secondary importance.

SUMMARY AND CONCLUSIONS

1. Prolapse of the efferent segment of bowel through a colostomy is rare.

2. The important etiological factors are the size of the defect in the anterior abdominal wall, the redundancy of the bowel distal to the colostomy, stenosis of the distal segment, and increased intra-abdominal pressure.

3. The prevention of prolapse begins with measures to control the size of the defect in the abdominal wall. The palliative treatment includes manual reduction of the prolapse and the use of support to prevent its recurrence. Radical treatment includes plastic procedures to decrease the size of the defect in the abdominal wall and immediate or delayed amputation of the prolapse in either one or two stages.

REFERENCES

1. Antonioli, G. M. Policlinico, 1936, 43: 630–638.
2. Bevis, E. M. Northwest M., 1927, 26: 213–215.
3. Bickham. Operative Surgery. Vol. 5, pp. 8–9. Philadelphia and London: W. B. Saunders Co., 1928.
4. Charbonnel, M. J. méd. Bordeaux, 1923, 95: 651.
5. Devine, H. B. Brit. M. J., 1935, 2: 1245–1249.
6. Firor, W. M., and Poth, E. J. Ann. Surg., 1941, 114: 663–671.
7. Gabriel, W. B., and Lloyd-Davies, O. V. Brit. J. Surg., 1935, 22: 520–538.
8. Hall, W. E. B. Ann. Surg., 1936, 104: 475–477.
9. Lahey, F. H., and Cattell, R. B. Am. J. Surg., 1935, 27: 201–213.
10. Loubat and Fontain. J. méd. Bordeaux, 1923, 95: 577.
11. Madranges, M. Arch. fr.-belg. chir., 1923, 26: 1171–1172.
12. Reid, M. R. N. England J. M., 1936, 215: 753–765.
13. Idem. Am. J. Surg., 1933, 20: 359–365.
14. Veal, J. R. Surg. Gyn. Obst., 1940, 71: 672–674.
15. Whipple, A. O. Surg. Gyn. Obst., 1940, 70: 257–260.

EXPERIMENTAL CRUSHING INJURY

Peripheral Circulatory Collapse and other Effects of Muscle Necrosis in the Rabbit

E. G. L. BYWATERS, M.B., B.S. (Lond.), M.R.C.P. (Lond.), M.R.C.S. (Eng.), and G. POPJAK, M.D. (Szeged), London, England

THE syndrome culminating in renal failure seen in air-raid casualties following prolonged compression beneath débris has been thought, on clinical grounds, to be the result of muscle necrosis (Bywaters and Beall, 1941). As soon as the picture had been clearly defined in man, we decided to reproduce the condition, if possible, experimentally. The method used at first, of which this paper is an account, was only partially successful: only one aspect of the human condition was reproduced. This failure to reproduce the full picture might possibly be accounted for by the fact that the muscles of the experimental animal used did not contain myohemoglobin. Although disappointed at first, we may have gained a distinct advantage in that the complex problem has been split for analysis into two natural parts: first, those effects that have in man been classified as circulatory collapse, and, second, the effects classified as renal impairment. Other methods have been developed to reproduce renal failure. This paper is confined to the study of a "shock" syndrome produced in the rabbit by muscle necrosis. A second paper will deal with the experimental production of renal failure following myohemoglobin injection.

In man almost any serious collapse resulting from burning, bleeding, cutting, crushing, hurting, freezing or frightening, may be given this label "shock," often to the stultification of detailed observation. In the realm of experiment even greater diversity of means is thought to result in this single end "shock." It should be recognized that each type of trauma produces specific results and that the clinical syndrome called "shock"—pallor, sweating, coldness, and a decreased arterial pulse—is only a part of many diverse processes set in motion by various types of trauma, conspicuous because common to all. The advantage, therefore, of experimental inquiry over clinical observation is that trauma can be produced by a single simple and controlled process, thus rendering analysis easier. A common method of producing experimental shock is to beat, with a blunt instrument, a limb under general anesthesia; this produces soft tissue damage, hemorrhage, and usually fractures as well, with concomitant fat embolism, besides introducing the added complication of a prolonged anesthesia. The mode of production of shock described here is limited to a single main causal factor, muscle necrosis: decrease of circulating blood volume can be produced and measured without hemorrhage or fracture, without pain, as far as this can be judged, and without, or with a minimum of, preliminary anesthesia. Finally, the results of the method are directly applicable to a similar condition in man.

From the Departments of Medicine and Pathology, British Postgraduate Medical School.

This work has been done during the tenure of a Beit Memorial Research Fellowship by the senior author and a British Council Scholarship by the junior author, with the assistance of an expenses grant from the Medical Research Council.

METHOD

Hollow rubber pressure tubing of 3.5 millimeter internal diameter and 7.5 millimeter external diameter was wound tightly around one or both thighs of rabbits between 1.5 and 2.5 kilograms, starting at the knee and winding upward as high as thought desirable. The ends were then fastened in a screw clip. There was no further traumatization other than this tight binding. The length of leg encircled was then measured medially and laterally, and the circumference was obtained by marking a strip of paper passed around the bound part at its middle. The volume of muscle included

in this cylinder was calculated by subtracting from the total volume the volume of (1) the rubber tubing in its stretched state (a factor of the measured circumference and the number of turns used) and (2) the bone beneath (a factor of the length of leg enclosed). The animal was left for any desired period, after which the rubber tubing was removed. This removal appeared to be quite painless. Anesthesia was not needed for the application, since this appeared to be as relatively painless as in man, but we found that light ether narcosis facilitated rapid application of the tubing in the standard manner: it was not prolonged for more than a minute, and had no evident effect on the animal.

The animals were kept on a standard diet, consisting of 100 grams daily of a mixture of bran (2 parts) and dried beet pulp (1 part) moistened with 250 cubic centimeters of water, and were allowed to drink water freely; they were housed in metabolism cages at a fairly even room temperature (about 20 degrees C.) maintained during the winter months by central heating; the urine was collected either 24-hourly (with a few drops of toluene) or for shorter periods; for most of the experiments the urine was expressed by pressure on the belly at the desired time. Blood was obtained from the ear veins: to obtain serum free from hemolysis it was necessary to centrifuge before coagulation.

Biochemical analyses were performed by the following methods: blood urea, plasma chloride, phosphate and protein (King, Haslewood, and Delory, 1937); serum potassium (King, Haslewood, Delory and Beall, 1942); carbon dioxide combining power (Van Slyke and Cullen, 1917); urine protein (King and Haslewood, 1936); urine creatine and creatinine (Folin, 1914); urine urea (classical urease and nesslerization method); urine hydrogen ion concentration . . . . by a capillary tube method, using the British Drug Houses 4–11 indicator . . . . and then more specific ranges such as given by bromcresol purple; hemoglobin by the photoelectric method described by Hill and Pincock, 1941. Blood pressure was measured on the central ear artery by the capsule described by Grant and Rothschild (1934), care being taken to ensure full dilatation of the vessels with heat. Changes in extracellular tissue fluid volume (thiocyanate space) have been followed in four experiments, the change in final serum concentration of injected sodium thiocyanate being used, estimated as described by Lavietes and associates (1937) except that the serum was withdrawn without oil: allowance was made for the amount excreted in the urine. Permeability was investigated by the intravenous injection of trypan blue, 1 or 2 per cent solution in normal saline being used, the dosage varying between 50 and 200 milligrams per kilogram.

Autopsy was performed immediately after the animal was killed by a blow at the back of the neck. Tissue fluid was collected from the leg after removal of the skin, and, in some instances, the amount of necrotic tissue was measured volumetrically after dissection. Heart blood was also taken and urine was collected. The adrenals and kidneys were dissected free and weighed. Tissues were fixed in 5 per cent formol-saline and 5 per cent formol-Zenker.

RESULTS

After some orientating experiments in which the effect of compression for various times and of a varying extent of muscle was observed, a procedure was standardized for subsequent work on muscle necrosis and renal function. This was a compression of 4 to 6 hours by 8 turns of the rubber tubing. Most of the results given below apply to such a procedure, but, as will be seen when the effects of a varying time and extent of compression are discussed, it does not seem to matter whether compression was maintained for anything from 4 to 12 hours; within these limits the result is very similar. Further, while, with the standardized procedure, similar results were obtained in general in each experiment, yet there was considerable individual variation in quantitative detail. We have therefore selected, for illustration, typical examples of each type of experiment.

During the period of compression, the animal remained well: the blood urea might rise or fall a few points within normal limits, the blood pressure remained constant; the hemoglobin might rise or fall slightly, perhaps due to leakage of plasma into, or from, the

Fig. 1. Rabbit post mortem showing extent of muscle necrosis following the standard procedure (8 turns of tubing applied for 5 hours). The rabbit was killed 7 days after release. Note the sharp border and small petechial hemorrhages. Most of the edema has by now been absorbed.

compressed area. There might be a diuresis during this period, when the urine became dilute and more alkaline. Only on rare occasions did a slight trace of albumin or creatine appear (for the first time) in this urine, doubtless due to leakage from the compressed area.

After release of the compression. 1. General observations. Immediately after release, the compressed muscle was smaller than the opposite side, cold, and relatively hard to touch. The leg below the lesion was usually normal. Soon, within an hour, the compressed muscle began to swell and become doughy; pitting on pressure was felt afterward, and, in the course of a day, spread downward to affect the limb below the lesion and upward toward the groin. The limb was paralyzed, and, as the animal hopped round, it was dragged behind. Usually the animal recovered sufficiently to eat and drink within 6 hours of release, but its appetite was seldom normal for the first day. Recovery of the limb was never complete as regards use, owing to atrophy, but edema had usually disappeared by 2 weeks: often there was atrophy, not only of the adductor group and the lower quarter of the quadriceps, but also of the calf muscles as well.

Animals compressed for a time shorter than 2 hours were less incapacitated, showed much less edema and were not paralyzed: there was practically no atrophy. Similarly, all changes were less marked in animals in which the extent of the compression had been less (e.g., 2 turns of rubber tubing instead of 8 as in the standard procedure). Animals compressed for 12 hour periods showed no greater change than did those compressed for periods of 4 hours. If, however, the extent of the damage was increased by repeating the standard compression on the opposite limb at the same time, or within a day, death occurred in "shock" with a low blood pressure. Degrees of damage intermediate between this and the standard procedure produced intermediate results. Rarely death occurred after compression of one limb, in which a greater quantity of muscle had been damaged than in the standard procedure. In 40 experiments in which one limb was crushed by the application of 8 to 10 turns of tubing (i.e., 1 to 2 milliliters of necrotic tissue per 100 grams body weight) for periods varying between 3 and 12 hours, but mostly of the standard time, 4 to 5 hours only 3 died. Details of these follow.

2. Pathology. Pathological changes were seen postmortem in the muscles; only slight changes in the kidneys and adrenals, and little elsewhere. In summary, there was subcutaneous edema in animals surviving several hours, extending from the toes up over the belly wall and into the rump, often extending as well into the opposite rump. The muscles were swollen and edematous, unusually pale and friable from the first or second day on, with a well marked line of demarcation (Fig. 1). There was no hemorrhage other than occasional petechiae, most numerous in the boundary zone between living and dead tissue. Necrosis occurred, in the majority of animals, only beneath the compressing band. A circulation adequate to supply the distal part of the limb is probably maintained through the femoral marrow spaces. This may explain why it is usually impossible to produce necrosis with a single ligature except immediately beneath it, unless the tubing is applied (as in Wilson and Roome's work in the dog) actually around the leg at the level of the hip joint. This procedure would minimize the circulation through the femur. In only 3 of 40 one-leg crush experiments was there necrosis distal to the compression (in the gastrocnemius, soleus, and anterior tibial muscles). Two of these 3 animals and 1 other rabbit with highly

Fig. 2.

Fig. 3.

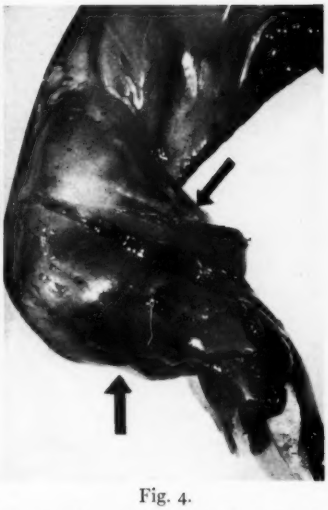


Fig. 4.

Fig. 2. Trypan blue injection in rabbit crushed by standard procedure. Note subcutaneous bluing and edema extending down to foot and up over reflected flap of abdominal skin.

Fig. 3. The same rabbit, with subcutaneous tissue removed from leg, showing muscle necrosis and bluing extending a small distance above and below the necrotic area.

Fig. 4. The same rabbit with muscle incised to show the sharp boundary between intense blue stained necrotic tissue and more normal edematous tissue nearby only lightly stained.

acidified urine and blood were the only ones that died following compression of 8 to 10 turns on one leg for 4 hours and upward. It is thought that vasospasm was responsible for this necrosis. Barnes and Trueta (personal communication) have demonstrated that this does occur in rabbits with single light wire ligatures round the thighs. In favor of this, as an explanation, was the character of the necrosis in the distal muscles, which was rather patchy, without any definite gross demarcation zone between living and dead tissue such as was seen beneath the compressing bands. In 7 experiments, trypan blue was injected intravenously to delineate the damaged area. In 1 rabbit weighing 1800 grams, into which 70 cubic centimeters of 2 per cent trypan blue in saline had been injected 6 minutes before release, the blue area seen at postmortem 40 minutes after release corresponded exactly with the area over which rubber tubing had been applied; above and below there was no increase in permeability. When the necrotic area was directly observed in an anesthetised animal injected with trypan blue immediately after release of the compression, the dye was seen in the large blood vessels running through the crushed area to the distal part of the leg within a few seconds of injection. The compressed area, white compared with the pink normal muscle proximally and distally, only gradually became distinctively colored: the blue appeared first surrounding the larger branches of muscle arteries at about 15 minutes after release, and was only distinctly visible over the whole area after 30 minutes. When injected later, for instance 20 minutes after release, the coloration was much less rapid. In the late stages of the lesion, for instance a week after release, the dye was most intense in the boundary zone between the normal muscle and the central white "infarct" of necrotic and coagulated fibers. At 20 hours after release of compression and simultaneous trypan blue injection, there was much blue dye not only in the necrotic area but in the subcutaneous edema fluid extending up over the abdomen into the rump and down to the toes (Figs. 2, 3 and 4). The fluid contained dye in a concentration equal to that in the plasma. The result of these injections was taken to indicate

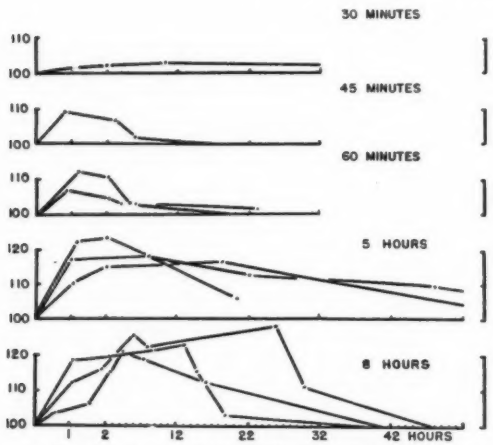


Fig. 5. Hemoconcentration following application of rubber tubing (8 turns) for varying periods: expressed as percentage rise from initial value.

a local increase in permeability of the vessels in the damaged area and lymphatic drainage of the protein-dye combination. No general bluing was seen other than in liver and renal cortex, thus presenting no evidence for a generalized increase in capillary permeability. In the acute stage the internal organs appeared dry; there was no hyperemia.

In animals allowed to recover, atrophy was pronounced: this sometimes affected the distal part of the limbs as well as the compressed area. It is thought that this was due to a nerve lesion, as no evidence of necrosis was seen distally in the majority of the experiments.

3. Hemoconcentration. Immediately after release, the hemoglobin percentage began to rise, due to loss of plasma into the damaged area. Figure 5 shows the time relation of this hemoconcentration. There was a rapid rise in the first hour to reach a plateau, which was usually maintained until between 10 and 20 hours. There might be a peak any time during the second or succeeding hours, usually between 1 and 4 hours after release. After this plateau the hemoglobin fell away to normal as the lost volume was restored. In 11 experiments (of a total of 21) in which a reading was made at 1 hour after release, rises of between 10 and 29 per cent were seen, with an average rise of 18 per cent; the same average figure

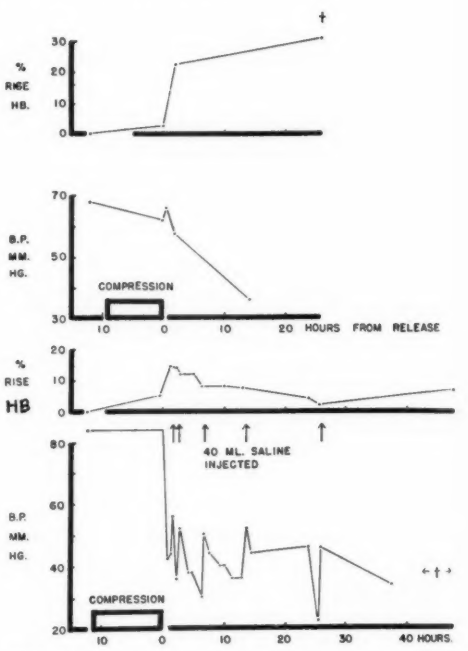


Fig. 6. Hemoconcentration (as percentage rise) and blood pressure fall (in mm. Hg.) in 2 rabbits with both legs compressed for 8 turns, one treated with 5 intravenous saline injections of 40 milliliters each. Death occurred in each at the point indicated by the cross.

was found at 1 hour in 7 experiments in which both legs were compressed (group B1).

If the compression was applied for about 8 to 10 turns on each leg, 7 of 7 untreated animals died between 4 and 26 hours after release. Even intravenous injection of saline (5 animals) or rabbit serum (4 animals) made no difference in the results, except in one animal which survived longer than usual and died between 42 and 48 hours. Figure 6 compares the hemoconcentration and blood pressure fall in this animal with the animal surviving longest of the untreated group. Saline given early, before hemoconcentration has reached a maximum, appeared to maintain blood volume at a higher level, as judged by hemoglobin concentration. If given later when hemoconcentration was well established, it made little difference. Despite the maintenance of blood volume, as judged by hemoglobin level, the blood pressure showed as

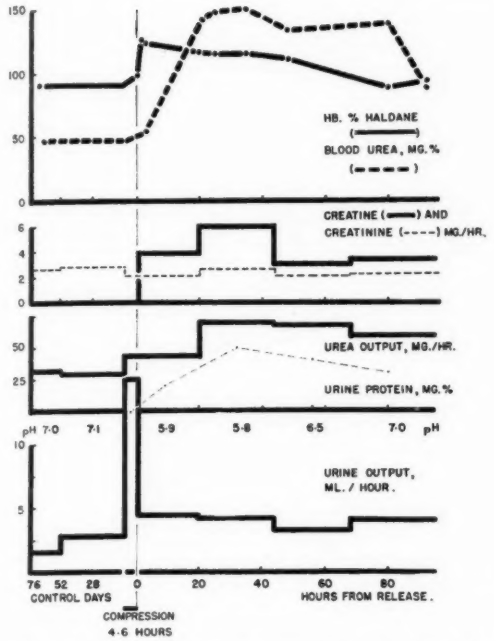


Fig. 7. Part of both thighs compressed for standard time: shock, although pronounced, was not severe enough to produce death. Note the acid urine, the rise in urea and hemoglobin, the increased output of urea and creatine and the maintenance of renal function.

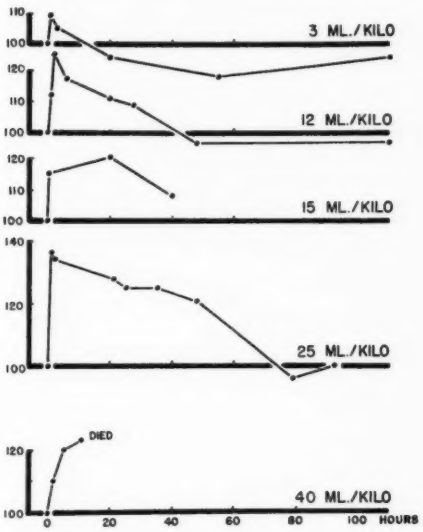


Fig. 8. Hemoconcentration (expressed as percentage rise from control value) in rabbits compressed to a varying extent for the standard time.

great a fall as in untreated animals, and death finally occurred. A rough analysis of the height and time of appearance of the peak hemoconcentration shows little difference between these fatal experiments (with an average calculated amount of necrotic tissue equal to 3 to 4 milliliters per 100 grams body weight) and those experiments in which one leg only was crushed (with an average calculated amount of necrotic tissue equal to 1 to 2 milliliters per 100 grams body weight). Similarly, hemoconcentration seems to bear no obvious relation to the mortality in the 4 experiments in which an intermediate amount of muscle was damaged (group C). Figure 7 shows one of these experiments, in which one leg was crushed in the routine way with 8 turns of rubber tubing, and the opposite leg bound with 3 turns only, resulting in an estimated amount of necrotic tissue equal to 2.4 milliliters per 100 grams body weight. (Actual volume measurements of the necrotic edematous tissue 4 days afterward gave a figure of 2.8 milliliters per 100 grams). It will be seen that the maximum hemoconcentration is +36 per cent; this is higher than the percentage reached by 11 of the whole fatal group in which both legs were compressed for 8 to 10 turns each.

On the other hand, in a group of 5 experiments (group D) in which the one leg was crushed to a lesser extent than normal, 2 to 4 turns, resulting in an estimated necrosis of 4 to 14 milliliters = 0.2–0.7 milliliters per 100 grams body weight, and an actual measured necrosis of 0.2–0.8 milliliters per 100 grams, the hemoconcentration tended to be smaller and proportional to the amount of tissue damaged, as may be seen in Figure 8. The peak tended to be earlier, between 1 and 2 hours, as well as lower.

In a similar way, hemoconcentration was small and limited, both in height and duration, in a further group of 8 experiments (group E) in which compression was applied to the full extent but for shorter periods, from 30 minutes to 2 hours. Some of these are illustrated in Figure 5.

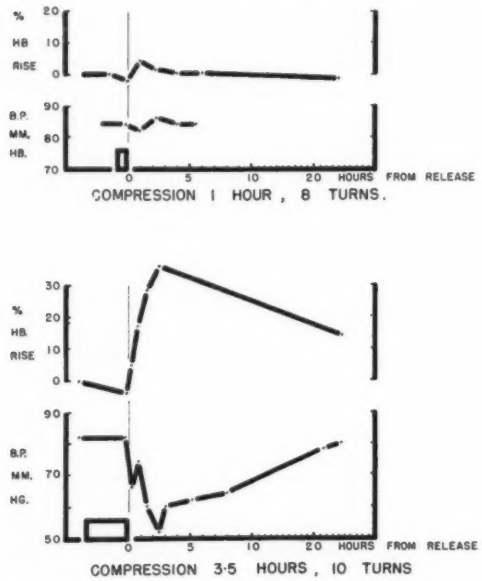


Fig. 9. The effect of a 1 hour period compared with that of a 3.5 hour period of compression on hemoglobin and blood pressure, with similar extent of injury.

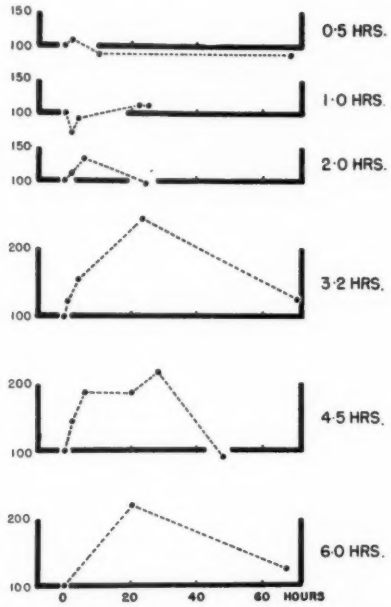


Fig. 10. Blood urea rise with compression for periods varying from 0.5 to 6 hours.

4. Blood pressure. The systolic pressure in the ear artery might fall rapidly after release, but was usually maintained until after hemoconcentration had occurred. With the standard procedure a moderate fall of 10 to 20 millimeters of mercury might occur which was held for about 20 to 30 hours. Not infrequently there was little or no fall despite the usual degree of hemoconcentration. There appears to be no direct correlation between the extent of the blood pressure fall and the degree of hemoconcentration; the duration of the two is better correlated.

With lesser amounts of damaged muscle, no fall of blood pressure occurred, corresponding to the very small hemoconcentration in such experiments (Fig. 9). With greater extent of necrosis, as after compression of both legs, there was usually a dramatic fall in blood pressure (Fig. 6). This could be partially restored toward normal with intravenous saline, but the beneficial effect was only very temporary. With 8 to 10 turns on each leg, there was a progressive fall of blood pressure until death occurred.

After compression for shorter periods than 3 hours, there was little or no fall in blood pressure; this appeared to occur only when hemoconcentration approached that point seen following the standard procedure (and as pointed out above, not always then).

5. Blood urea. The blood urea concentration rose after release of the compression; but not as rapidly as did hemoglobin. The peak concentration occurred between 20 and 40 hours after release; from a normal level in the rabbit with unrestricted water intake of about 25 to 45 milligrams per cent and in rabbits kept on restricted fluid intake of 30 to 60 milligrams per cent the blood urea rose, following the standard procedure, to a maximum of between 50 and 150 milligrams per cent, averaging about 100 milligrams per cent. It then fell away more slowly than it rose (Fig. 10).

As will be detailed later, there was no obvious failure of the renal tubules in concentrating urea, and the urinary urea output was usually greater than in the control period; we must therefore ascribe this increase in blood urea at least in part to increased tissue break-

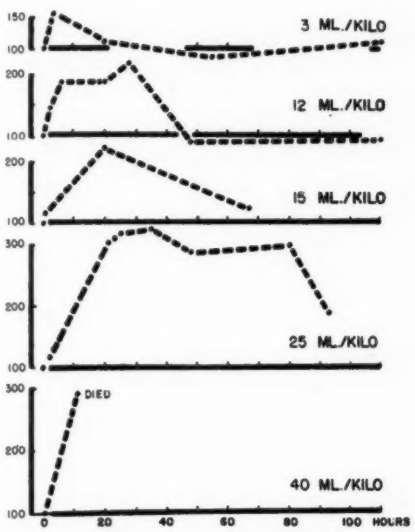


Fig. 11. Blood urea rise with compression of varying extent for the standard time.

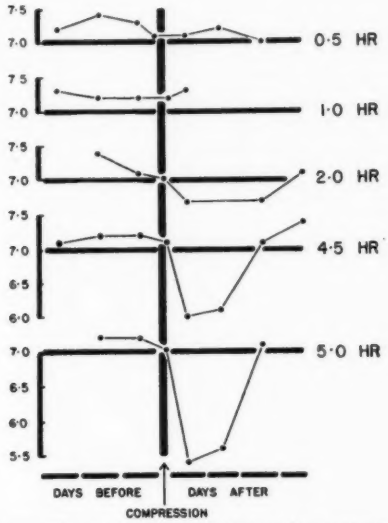


Fig. 12. Change in urinary hydrogen-ion concentration following compression for periods varying from 0.5 to 5 hours.

down. Other factors concerned may be decreased renal blood flow and decreased filtration pressure, particularly in the rabbits dying with low blood pressure after compression of both legs.

An inspection of Figures 11 and 12 which illustrate the course of blood urea determination in groups B, C, D (with varying extent and duration of crush) shows a similarity in behavior to that of hemoglobin values, except that the blood urea is less responsive to crushes of small extent.

6. Other biochemical changes in the blood. a. *Serum potassium.* Rabbit blood is very susceptible to hemolysis: should this more than tinge the serum pink, the potassium content may become erroneously high. We found it necessary to centrifuge the blood before coagulation had occurred. A further source of error was that blood drawn from the heart immediately after killing the animal by a blow on the neck also gave erroneously high values. Failure to appreciate this point vitiated many early observations. After rejection of all estimations taken on blood at death and all specimens with traces of hemolysis, it was found that following crush the potassium content did not rise above normal (15 to 23 milligrams per cent, average of 32 values 19 milligrams per cent) in the majority of animals. One experiment only showed a rise, from 20 milligrams per cent before to 28 milligrams per cent at 48 hours, falling to 22 milligrams per cent at 120 hours. The 13 remaining experiments included 2 experiments in which both legs were crushed, with a calculated necrosis of 25 milliliters per kilogram, and one experiment in which only 2 turns were made, with a calculated necrosis of 3 milliliters per kilogram. The remainder followed the standard procedure. This may be compared with the potassium rise following simple ligature of both ureters (e.g., from 21 milligrams per cent before to 30 milligrams per cent on the second day).

b. Phosphate. This was not increased from the normal level of 2.5 to 6.1 milligrams per cent (average of 33 values 4.5 milligrams), in animals crushed by the standard procedure, or in animals in which both legs were crushed up to 25 milliliters per kilogram. The higher values within the normal range did not appear to be correlated with high blood urea rise or high potassium figures. For contrast, in rabbits with ligated ureters, the phosphate rose parallel with the urea and potassium

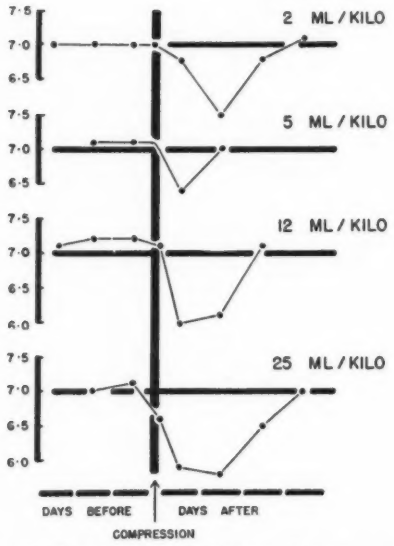


Fig. 13. Change in urinary hydrogen-ion concentration following compression of varying extent for the standard time.

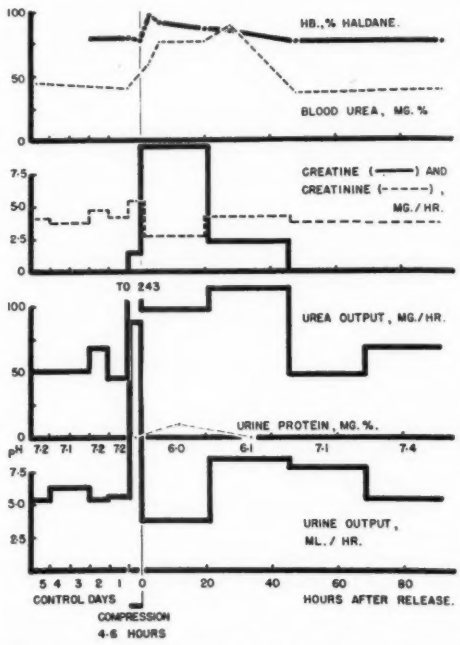


Fig. 14. Changes following the standardized compression (one leg, 8 turns for 4.6 hours). Note blood urea and hemoglobin rise, creatinuria, increased urea output, and acidity. No renal impairment.

(e.g., from a normal level of 4.6 milligrams per cent to 6.5 and 8.9 milligrams per cent on the 2d and 4th postoperative days respectively. This paralleled a urea rise to 430 milligrams per cent and a potassium rise to 30 milligrams per cent).

In this estimation again, hemolysis must be avoided: specimens taken from the heart immediately after death may occasionally show abnormally high results, and all such estimations have therefore been discarded.

c. Plasma protein. From an average control figure of 6.5 grams per cent (33 normal values ranging from 5.8 to 8.0 grams) the total plasma protein concentration fell 5 to 10 per cent from the initial value, reaching a minimum between 2 and 4 hours after release, in general therefore running inversely parallel to the hemoglobin concentration. It then returned to the precompression figure within 24 hours. (Characteristic examples are seen in Figs. 18, 19 and 20). This behavior is opposite to that of hemoglobin: the explanation we suggest is that the blood loses protein into the damaged area and gains water from the tissue spaces faster than the reserve protein can be mobilized.

d. Carbon dioxide combining power. From an average control figure of 42 milliliters per 100 cubic centimeters (17 normal values ranging from 34 to 57 milliliters per cent, the alkali reserve fell soon after release (2 to 4 hours) to 40 to 80 per cent of the control figure, often returning to normal values within 24 hours (Fig. 19), sometimes remaining low for a longer period (Fig. 18).

7. The urine. a. Formed elements. The first urine passed after release of the compression usually contained hyaline casts. These might be numerous; they gradually disappeared during the next few days and were replaced by a few granular casts. In the later specimens, sometimes granular casts containing small cuboidal epithelial cells were seen; on occasion, these small cuboidal epithelial cells appeared free and were numerous. It is not thought that these came from the bladder. There were no red corpuscles and the benzidine test was negative.

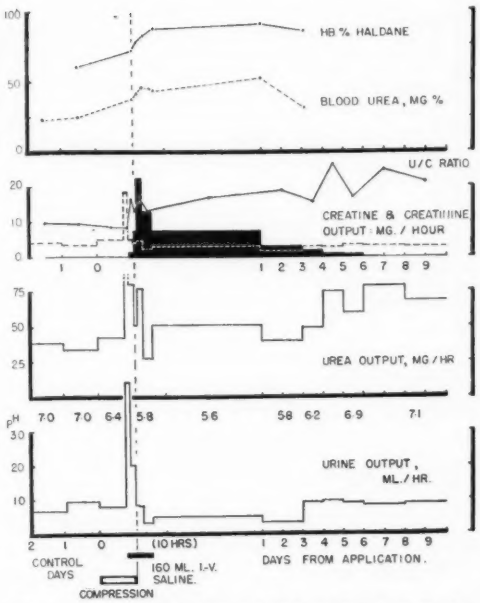


Fig. 15. Changes in the period immediately following release from a standardized compression, observed with a self-retaining catheter *in situ* for 4 hours. Saline given intravenously, as indicated, to maintain flow during development of shock. Note increase in urea creatinine ratio, immediate appearance of creatine after release and pre-release diuresis.

b. Pigment and protein. There was no pigment passed that could be detected spectroscopically or by the benzidine test. The first and succeeding specimens usually contained protein, usually between 10 and 50 milligrams per cent. On 2 rabbits there was none passed, despite a high degree of hemoconcentration (to 153 per cent and to 146 per cent of the pre-operative level) and of blood urea retention (up to 104 and 190 milligrams per cent respectively). The latter rabbit had had 2 legs crushed on successive days. Other experiments in which an increased quantity of muscle was damaged showed the usual quantity of protein. Compression for shorter periods than the standard time showed less protein—none for ½ hour, none or 10 milligrams per cent in 1 specimen for 1 hour crushes, none or up to 20 milligrams per cent for 2 hour crushes. Compression of a lesser extent than the standard showed no constant reduction of protein.

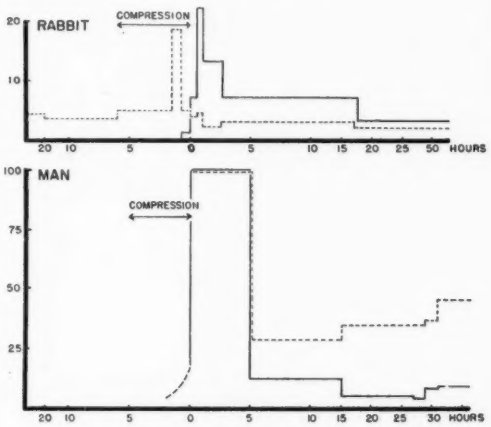


Fig. 16. Comparison of creatine, ——, and creatinine, – – – –, excretion in man and rabbit following crushing injury.

c. Urinary acidity. This is of importance with regard to its probable rôle in the precipitation of urinary hemoglobin as acid hematin following mismatched transfusion in man (Baker and Dodds). The urine became highly acid soon after the compression was released; the hydrogen-ion concentration fell from a

TABLE SHOWING RELATION BETWEEN (a) EXTENT OF NECROSIS IN 11 RABBITS FOLLOWING APPLICATION OF COMPRESSION FOR 4·5 TO 5·5 HOURS AND (b) THE PERCENTAGE INCREASE IN UREA OUTPUT OVER 3 DAYS, COMPARED WITH 3 DAYS CONTROL OUTPUT BEFORE COMPRESSION

Weight—kgm.	Calculated necrotic muscle—ml.	Necrosis—ml. per kgm.	Max. blood urea—mg.	Per cent change in urea output
1.89	4.0	2.1	45	—4
2.38	7.1	3.0	49	+3
1.61	9.2	5.7	41	+50
1.59	19.3	12.1	35	+56
1.97	21.6	11.0	52	+106
2.27	22.0	10.0	104	+34
1.34	23.6	17.6	37	+21
2.07	24.7	12.0	87	+30
1.66	30.7	23.5	150	+111
2.01	31.6	15.7	52	+77*
1.65	34.3	20.7	118	+7

*Control period 2 days only.

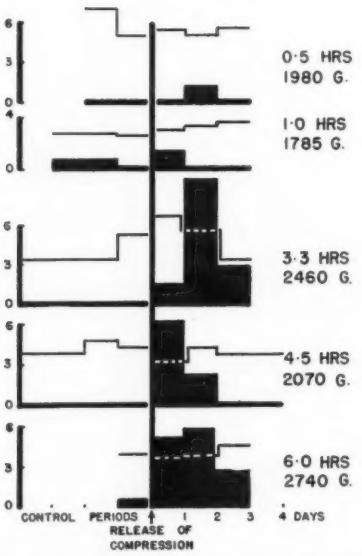


Fig. 17. Creatine and creatinine output after release of compression for periods varying from 0.5 to 6 hours.

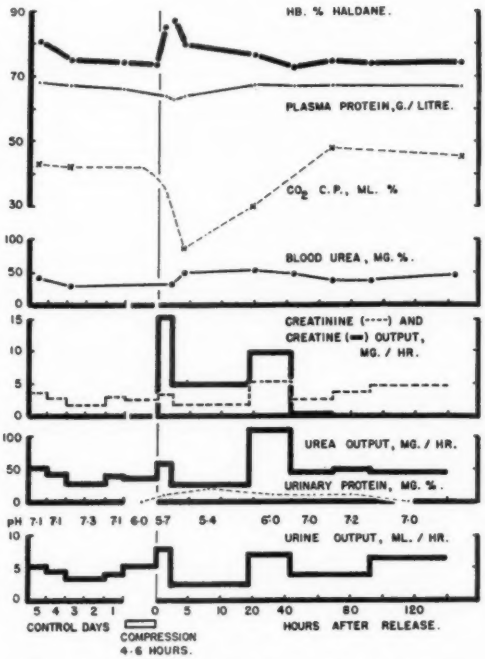

HB. % HALDANE.
PLASMA PROTEIN, G./ LITRE.
CO_2 C.P., ML. %
BLOOD UREA, MG. %.
CREATININE (----) AND CREATINE (—) OUTPUT, MG. / HR.
UREA OUTPUT, MG. / HR.
URINARY PROTEIN, MG. %.
URINE OUTPUT, ML. / HR.
CONTROL DAYS
COMPRESSION 4·6 HOURS.
HOURS AFTER RELEASE.

Fig. 18. Changes following the standardized compression (8 turns for 4.6 hours). Note the fall in carbon dioxide combining power within 4 hours of release.

normal level of 7.0–7.2 to a minimum of 5.4. With the standard procedure the minimum lay between hydrogen-ion concentration 5.4 and 6.0; the lowest value usually occurred between 5 and 20 hours after release. Neutrality was regained by 2–3 days after release, sometimes earlier (see Figs. 12 and 13).

d. Urinary urea and creatinine. There was usually an increase in the output of urea following the crush, often to double the previous level (Fig. 14). The creatinine output remained much more constant, and therefore, as can be seen in Figure 15, the urea creatinine ratio in the urine rises. During this time the rabbit usually ingested a normal amount of food protein, except sometimes during the first 24 hours. The conclusion to be drawn is that there was increased protein breakdown. Judged by excretion this was maximal on the first or second day. Several cases analyzed more closely showed an immediate increase in output of urea, before release, due to diuresis (Fig. 14). The urea-creatinine ratio stayed constant. Immediately after release (or before, if leakage occurred, as indicated by the appearance of creatinuria), the urea-creatinine ratio increased, due to a rise in the total urea output. This might remain up for a week. The extent and duration of the increased output of urea bore little if any quantitative relation to the extent of the damage (Table I). No evidence is presented on the source of this protein breakdown, whether local or general, but according to Cuthbertson, both mechanisms are involved in similar experimental preparations.

e. Urinary creatine. This appeared in the urine immediately after release (Fig. 16). As may be seen from Figure 17, the amount liberated was small below 2 to 3 hours' compression, while above this point no further significant increase was seen with increasing duration of compression. In a similar way, no definite relation was seen between amount of tissue made necrotic and creatine set free.

8. Tissue fluid. The edema fluid protein content at postmortem in 21 of the experiments varied between 2.4 grams and 4.8 grams per cent, with an average of 3.6 grams per

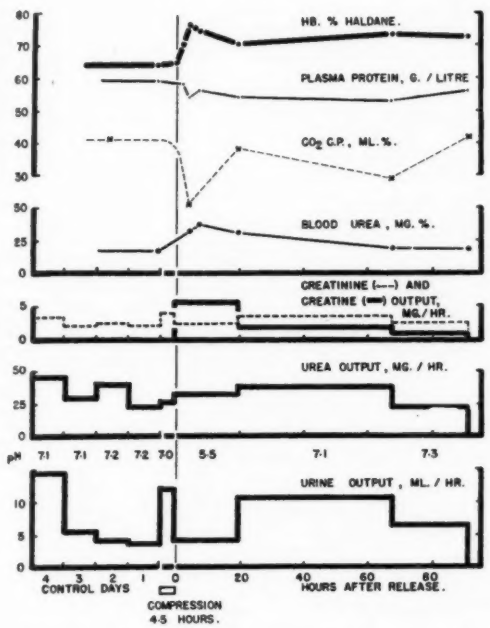


Fig. 19. Changes following the standard compression (8 turns for 4.5 hours). Note the fall in plasma protein and the increased urine output on the second and third days.

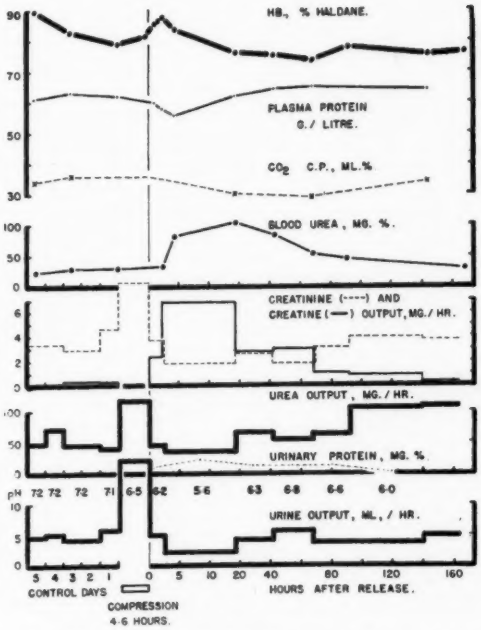

HB., % HALDANE.
PLASMA PROTEIN
G./ LITRE.
CO_2 C.P., ML.%
BLOOD UREA, MG. %.
CREATININE (----) AND
CREATINE (——) OUTPUT, MG./ HR.
UREA OUTPUT, MG./ HR.
URINARY PROTEIN, MG. %
pH 7·2 7·2 7·2 7·1 6·5 6·2 5·6 6·3 6·8 6·6 6·0
URINE OUTPUT, ML. / HR.
5 4 3 2 1 0 5 10 20 40 80 120 160
CONTROL DAYS
COMPRESSION
4·6 HOURS.
HOURS AFTER RELEASE.

Fig. 20. Changes following the standard compression (8 turns for 4.6 hours). Note the delayed increase in urea output.

cent. Expressed as a percentage of the terminal plasma protein in 20 of these experiments, it ranged from 46 to 77 per cent with an average of 59 per cent of the plasma value.

The "thiocyanate space" which is a measure of the extracellular tissue fluid volume, showed some slight increase following release of the compression in a few preliminary experiments. The increase, however, was usually small (Fig. 21) and could be accounted for by the volume of cellular material rendered permeable to thiocyanate by necrosis. This would mean that there was no cellular dehydration with that degree of damage. Equilibrium, which is reached between 30 and 60 minutes, following injection in the normal, required a longer period after following release of compression.

ANALYSIS OF STUDY

The experimental lesion. The findings indicate that the experimental injury sets in motion two main processes: (a) a loss of substances (fluid, protein and salt) from the bloodstream and from the body generally into the injured part, causing first, *local increase* in fluid, protein and salt, manifest as edema, and second, *general decrease* in fluid, protein and salt; manifest as hemoconcentration, fall in plasma protein and tissue dehydration.

(b) An accession of substances from the damaged area into the bloodstream (directly and via the lymphatics) and thence into the body generally and into the urine. Some of these substances seem to be of an acid nature, as manifest by the fall in carbon dioxide combining power of the plasma and the fall in urinary hydrogen-ion concentration: it seems possible that one of these substances is lactic acid.

Another of the substances found in the urine which is derived from the damaged muscle is creatine. This occurs under any conditions in which muscle fibers undergo autolysis. Whether potassium, phosphorus, amino acids, or other easily stored products of cellular

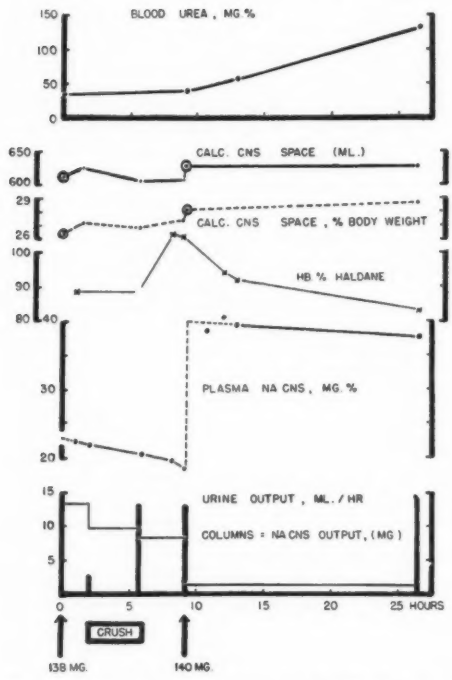


Fig. 21. Changes in thiocyanate distribution space (extracellular tissue fluid volume) following compression. Circles indicate volume calculated by extrapolation following injection; dots indicate volume calculated after subtraction of thiocyanate excreted in the urine. Note the very small rise (from 613 to 628 ml. only), which is less even than the amount of cellular tissue rendered permeable to thiocyanate by necrosis (75% of 30 ml., the total volume of tissue compressed).

autolysis are also liberated, has not yet been investigated. The potassium and phosphorus concentration in the blood serum do not rise beyond normal limits after release of the compression.

Relation to human types of injury. The secondary effects of the loss of fluid protein and salt into the damaged area, classified under (a) e.g., hemoconcentration, fall in blood volume, vasoconstriction, and fall in blood pressure, are described, when seen in man, as "shock." As we have recently learned however, hemoconcentration in human "shock" is rare and occurs chiefly following burns and crushing injury (Grant and Reeve, 1941). It is due to loss of plasma into the damaged area. This method, therefore, seems suitable for reproducing in the rabbit these particular aspects of burns or crushing injury known, loosely, as "shock."

Furthermore, the effects classified under (b) (due to substances leaving the damaged area) are also seen in man following crushing injury. The urine is very acid, the plasma carbon dioxide combining power is diminished and creatine occurs in the urine. The resemblance of the "crush" syndrome in man to that produced in the rabbit by this method is thus very close. In two particulars, however, the parallel fails: the injury in man is accompanied by loss of myohemoglobin from the damaged muscle which comes to resemble fish flesh, and by its excretion in the urine; this may be followed in a few days by signs of renal failure (oliguria, increase of blood urea, phosphate, creatinuria, and decrease in carbon dioxide combining power). In two-thirds of the reported cases, death has occurred (Bywaters, 1942). The failure to produce myohemoglobinuria and the inability to produce a renal lesion severe enough to cause death, or indeed any serious degree of uremia, are, we think, associated both with each other and with the absence of myohemoglobin in those muscles of the rabbit which we compress. This has led us to another line of inquiry, which will form the subject matter of another paper. However, we think it is of interest that we should be able to reproduce the "shock" aspect of crushing injury uncomplicated by any marked degree of renal damage.

Excretory function. Excretory impairment is manifest in the rabbit in the first 24 hours after release, as judged by the small rise in blood urea and the decrease in urine volume and urea and creatinine output sometimes seen. In most of the experiments, however, the urea output was increased over that of the control period; this, we thought, was due to increased protein breakdown, which can be considered as partly responsible for the rise in blood level. The concentration of urea in the urine was fairly high during the oliguric period, often exceeding 2 grams per 100 milliliters, and we attribute this temporary failure of excretion, therefore, not to renal impairment, but to increased formation of excretable substances and to the reduced circulating

blood volume. It seems possible that if the blood volume be diminished due to loss of plasma, the venous return will be less, the cardiac output less, and the renal blood flow may therefore be diminished.

Some slight renal damage, however, does occur, if proteinuria and cylindruria be taken as sufficient evidence. However, both these occurrences are seen in man without evidence of a renal lesion (postural or orthostatic albuminuria and the albuminuria and casts seen after exercise); their sudden appearance in the rabbit following release of compression might indicate an increased permeability of the glomerular tuft or some lesion of the tubules. There is no evidence of an increase in capillary permeability other than in the injured area, as judged by trypan blue coloration, but it is possible that the glomerular tufts are particularly sensitive. An alternate possibility suggested by Eggleton (personal communication) is the protein in the urine might be the first stage breakdown of muscle protein, molecules small enough to be filtered by undamaged glomeruli. This has not yet been investigated. Summarizing, renal damage, if it exists at all, is very slight and of short duration. In this, the impairment of excretory function is unlike that seen following crushing injury in man, but resembles that seen following other experimental injuries designed to induce a 'shock'-like condition, certain relevant types of which will be considered next.

Other experimental lesions. Many able reviews of the experimental methods used to produce 'shock'-like conditions have recently appeared (Harkins, Moon, Blalock) and it is only necessary here, therefore, to mention those types of injury whose effects resemble those we have produced in the rabbit and those seen in the crush syndrome in man. Those effects seen in crushing injury in man and in the rabbit, classified as due to substances leaving the blood stream, are common to the injury of burns, to that following the injection of concentrated salt or glucose solutions underneath the skin, intraperitoneally, with or without subsequent removal of the effusion, or intravenously with salt and water loss in the urine, following necrosis of bowel or intraperitoneal implantation of minced muscle. Minor but important differences distinguish the results of these varying procedures; these appear to be due chiefly to such variables as the reavailability of the substances so lost, the rate of loss, and the total supply of sodium chloride or water available to the body, and the presence or absence of secondary infection (e.g., anaerobes in the peritoneum). When excretory function has been investigated, it appears in most of these experimental lesions to be impaired only temporarily, and is demonstrated either by such phenomena as albuminuria and cylindruria, or, if a severe strain is placed on the kidneys as, for instance, in Black's (1940) experiments with an increased urea load, by a rise in blood urea. In common with the conditions mentioned, excretory impairment in this experimental crushing injury appears to be temporary, functional rather than structural in nature, and dependent on such "extrarenal" causal factors as: (1) increased urea formation, (shown by the increased urea output with a normal creatinine output); (2) tissue dehydration (shown by general dryness and by the accumulation of fluid locally without gain in weight of the animals, together with a fall in serum protein and rise in hemoglobin concentration); (3) fall in filtration pressure in some cases, due to fall of blood pressure; and (4) perhaps to decreased renal flow (as a possible result of oligemia) and other factors yet unknown.

The effects in the rabbit corresponding to those in man which were here classified as due to substances leaving the damaged area, e.g., the acidosis and the creatinuria, have been reproduced by methods more closely resembling our own.

Andrews (1927) in the course of experiments designed to study the effect of acidosis on colloid structure and water metabolism, observed that an acidosis developed in dogs whose paws had been tied too tightly to the operating table, as soon as the ligature was released. He was unable to identify the acid, but thought it might be lactic acid. Apparently all his animals received large quantities of 5 per cent sodium chloride intravenously, and "nearly half" showed a picture like that

of uremic coma. Unfortunately, the description of methods and results is too inadequate to draw any useful conclusions from them. A similar procedure was adopted by Wilson and Roome in 1936. They studied with great care a series of 19 dogs in which one hind limb had been ligated under sodium barbital anesthesia by two turns of rubber tubing wound round the legs at the level of the hip joint. A metal skewer was used to keep the tourniquet at this level. Two animals constricted for less than 3 hours recovered, as did 4 of the remaining 17 constricted for longer periods. Radio-opaque injection showed no arterial spasm; death could be prevented by amputation and transfusion but by neither alone. Observations on the presence of clostridia in the muscle were made, but no deductions were possible as to the rôle they played, as they occurred also in the control limb. However, gas bubbles and crepitations were noticed in 3 animals. It seems that the lesion produced by Wilson and Roome was very similar to our own: it was complicated, however, by a prolonged barbiturate anesthesia, with inanition (which by itself killed 1 of 6 controls), and by the presence of an open wound and a Welch-like infection. They concluded that the amount of fluid lost into the ligated limb was adequate to cause death, in conjunction with the prolonged anesthesia and inanition. A somewhat similar study in 60 three month old rats was undertaken by Salzburg and Kabat (1941). They were interested mainly in the difference in survival (following ischemia) of normal and neoplastic tissue; no biochemical data are given. They found that if the circulation to the hind limb was cut off by rubber ligature, gangrene ensued. If, however, ischemia was produced by an inflatable rubber cuff at a temperature of 82 degrees F., the rats all survived a seven hour stasis, but none survived ischemia maintained for 8 hours. An inflammatory edema and some atrophy was noticed in the surviving rats. Allen's (1938) work on the survival of ischemic muscle at various temperatures is also relevant. He found that tissue survived much longer periods of ischemia if cooled; the critical ischemic time was decreased if the temperature was raised. More recently, Cuthbertson (1942), having followed up a study in man by one of metabolic changes following fractures in rats, has laid considerable emphasis on the general increase in metabolism which accompanies the local metabolism of trauma; he suggests that the local tissue loss is inadequate to account fully for the increased nitrogen, potassium, and phosphate wastage. This is a possibility which we have not yet investigated.

It would be of very great interest to investigate the effects of a similar injury in an animal, such as the dog, whose muscles contain myohemoglobin. Some work of this nature is already in progress (Winton and Eggleton, personal communication): renal damage and myohemoglobinuria has been produced.

CONCLUSIONS

Few conclusions can be drawn except that this method of injury produced the results here described, that they are similar to the effects of crushing injury in man, except that myohemoglobinuria and renal failure do not occur. Other conclusions relevant to the crush syndrome in man, which may be useful in prognosis and the critical assessment of treatment, are:

1. There appears to be a minimum time below which ischemia does not produce necrosis; above this, prolongation of compression does not increase the severity of the lesion.

2. The severity of the lesion, that is the extent of muscle that has been damaged, is indicated within certain limits by the height and duration of the rise in blood urea and hemoglobin and the fall in urinary hydrogen-ion concentration and plasma carbon dioxide combining power.

SUMMARY

A method of producing graded injury in the rabbit is described: it consists of winding a rubber band round the leg so as to produce an ischemia which is maintained for a period of between 4 and 5 hours. The resulting course is characterized by hemoconcentration and loss of plasma into the injured area which becomes swollen and doughy. Azotemia occurs, which is thought to be due in part to the depressed circulation, but mostly to increased

tissue breakdown. Other manifestations of the latter are creatinuria and a depression of the carbon dioxide combining power of the plasma, associated with an increased urinary acidity. No impairment of renal functional ability occurs (as measured by the power to concentrate urea) that is grossly obvious, although a few hyaline and granular casts appear, and are sometimes accompanied by a trace of proteinuria. No myohemoglobinuria occurs; this is not surprising, as the compressed muscles contain practically none of this pigment. The condition differs from the human condition of "crush syndrome" only in these two respects (absence of renal failure and absence of myohemoglobinuria), and as there is ample anatomical reason in the rabbit for myohemoglobinuria to be absent, it seems possible that these two negative "occurrences" may be causally related to each other.

ADDENDUM. Since this paper was prepared for publication, an article by Duncan and Blalock (Ann. Surg. 1942, 115; 684) has appeared: they produced a similar lesion in dogs by compression between two rigid boards. The animals were not allowed to survive for long enough to investigate renal changes. "Shock" could be partially prevented by the application of a pneumatic sleeve, at a pressure of 40 millimeters mercury. As no figure is given of the nonprotein nitrogen rise in the treated animals, it is impossible on these data to make deductions as to the effect on renal function. It may be that the shock aspect of crushing injury could be treated more cheaply by bandages or cuffs (Patey and Robertson, Brit. M. J., 1942, 2: 53) than by plasma infusion, but there is yet no evidence whether these procedures would increase or decrease the mortality of this injury, which is always due to renal dysfunction, not shock, except in neglected patients.

REFERENCES

ALLEN, F. M. Surg. Gyn. Obst., 1938, 67: 746.
ANDREWS, E. Arch. Int. M., 1927, 40: 548.
BAKER, S. L., and DODDS, E. C. Brit. J. Exp. Path., 1925, 6: 247.
BARNS and TRUETA. Personal communication.
BLACK, D. A. K. Lancet, 1940, 2: 618.
BYWATERS, E. G. L. Proc. Roy. Soc. Med., 1942, 35: 321.
BYWATERS, E. G. L., and BEALL, D. Brit. M. J., 1941, 1: 427.
CUTHBERTSON, D. P. Lancet, 1942, 1: 433.
EGGLETON, M. G. Personal communication.
FOLIN, O. J. Biol. Chem., 1914, 17: 469.
GRANT, R. T., and ROTHSCHILD, P. J. Physiol., 1934, 81: 265.
GRANT, R. T., and REEVE, E. B. Brit. M. J., 1941, 2: 293.
HARKINS, H. N. Surgery, 1941, 9: 231, 447, 607.
HILL, D. K., and PINCOCK, A. C. Lancet, 1941, 2: 754.
KING, E. J., and HASLEWOOD, G. A. D. Lancet, 1936, 2: 1153.
KING, E. J., HASLEWOOD, G. A. D., and DELORY, G. E. Lancet, 1937, 1: 886.
KING, E. J., HASLEWOOD, G. A. D., DELORY, G. E., and BEALL, D. Lancet, 1942, 1: 207.
LAVIETES, P. H., BOURDILLON, J., and KLINGHOFFER, K. A. J. Clin. Invest., 1936, 15: 261.
MOON, V. H. Am. J. Clin. Path., 1941, 2: 361.
SALZBURG, P., and KABAT, H. Arch. Surg., 1941, 42: 917.
VAN SLYKE, D. D., and CULLEN, G. E. J. Biol. Chem., 1917, 30: 289.
WILSON, H., and ROOME, N. W. Arch. Surg., 1936, 32: 334.
WINTON, F. R., and EGGLETON, M. G. Personal communication.

ROUTINE ABDOMINAL PANHYSTERECTOMY

As Prophylaxis Against Cancer of Cervical Stump

KARL H. MARTZLOFF, M.D., F.A.C.S., Portland, Oregon

THE importance of a residual cervical stump, following supravaginal hysterectomy, as a possible nidus for subsequent cancer development has given rise to varying points of view and consequently to different practices.

The major issue seems to be whether, when abdominal hysterectomy is done for benign disease of the uterus or uterus and adnexa, total hysterectomy should be employed as an inflexible routine in order to forestall by one procedure all danger of future malignant change which presumably exists as long as there is the slightest cervical residuum.

Other issues which have been raised, such as removal of a symptom producing cervix as against later treatment of such a cervix if left as the result of a supracervical hysterectomy; the question of vaginal shortening; alteration in vaginal secretion; etc., are purposely omitted.

One cannot help but conclude from the recurring discussions and a perusal of the current literature that there is still confusion and misinterpretation of statistics by some as to this problem.

It becomes essential, therefore, from the standpoint of statistics if one desires to compare cervical cancer and uterine myomas, for example, to have clearly in mind whether one is comparing the incidence of cancer with myoma, the incidence of myoma with cancer, or, the simultaneous occurrence of myoma and cancer. Likewise, it is one thing to speak of the incidence of cancer of the cervical stump in relation to cervical cancer, and quite another to speak of the incidence of cervical cancer among cervices left as residua following supravaginal hysterectomy. Also, one is confronted by the problem when to classify a cancer occurring in a residual cervical stump as one that was in existence at the time of the original supravaginal hysterectomy or one that developed in a previously noncancerous "cervical stump," i.e., a *bona fide* "cervical stump" cancer. Therefore, the ratio of cervical stump cancer to cervical cancer in general is not particularly pertinent to this specific problem unless one knows the ratio of cervical stump cancer to all supravaginal hysterectomies. The latter again may be misleading if one does not know the incidence of unsuspected cervical cancer present at the time of the supravaginal hysterectomy. One sees, nevertheless, the incidence of cervical stump cancer among cervical cancers in general interchangeably quoted as representing the incidence of cancer in cervices remaining after subtotal hysterectomy.

From the Department of Surgery, University of Oregon Medical School. Read at a meeting of the Johns Hopkins Medical and Surgical Association, Baltimore, Maryland, February 21, 1942.

RELATIONSHIP OF "CERVICAL STUMP CANCER" TO CERVICAL CANCER

Table I reveals the fluctuations that occur in some of the reports that deal with the incidence of cancer of the stump in relation to cervical cancer in general. In addition, we should mention the experience of Pehan and Amreich at the first University Frauenklinik, Vienna. They encountered 13 cancers of the cervical stump during a 16 year period, comprising probably less than 2 per cent of the cervical cancers observed during that time.

From the foregoing it may be assumed that cancer occurring in the cervical stump following supravaginal hysterectomy comprises from 2 to 4 per cent of cervical carcinomas. There are a few statistics that give both higher and lower values than those here given. It is a problem to know how to evaluate them when one considers as will be shown how frequently supravaginal hysterectomy has been done in the presence of unsuspected cervical cancer in this special group of patients.

The difficulty of evaluating some of the statistics relating to this matter when presented in tabulation, can be appreciated when it is recognized that in numerous instances they are not comparable. Some authors in reporting the ratio of cervical stump cancer to cervical cancer include all stump cancers irrespective of how soon they manifest themselves after supravaginal hysterectomy. Others regard a 1 year postoperative period of freedom from "stump cancer" as evidence that the cancer did not exist at time of original operation; others believe that longer periods should be used as criteria to exclude the presence of unsuspected cervical cancer at time of supravaginal hysterectomy. Nevertheless, one generally sees figures—arrived at so differently—indiscriminately compared. For example:

Three of Laborde and Roques' patients, who had had hysteromyomectomies, developed cancer within a few months after their operations. Five of the 10 cervical stump cancers reported by Scheffey developed symptoms of vaginal bleeding within less than a year after supracervical hysterectomy. They may reasonably be assumed to have had unsuspected cervical cancer at the time of the first operation. Similarly, 29 (43 per cent) of 67 cervical stump cancers studied from this point of view by Behney probably had cancer at the time of the supravaginal hysterectomy. Twenty-three (47 per cent) of the 49 cancers cited by Meigs developed within 1 year after abdominal hysterectomy; while 4 of the 7 cases reported by Hochman evidently had unsuspected cancer at the time of operation. Similarly, the "majority" of the 21 cases of Johnson and Tyrone developed cancer within 1 to 2 years after supravaginal hysterectomy. Several of the cases reported by Faehndrich, Healy and Arneson, and Kretzschmar and Gardiner, fall reasonably under the same suspicion although they have utilized a 1 year period of freedom from stump cancer as signifying the absence of unsuspected cancer at the time of hysterectomy. Bowers also reports 16 cervical stump cancers, of which 10 (62 per cent) were unrecognized but existent at the time of operation, while Leonard, in a compilation of 56 cases of "stump" cancer in 1913 gave detailed information about 36, of whom 15 (41 per cent) probably had coexisting but unsuspected cancer when the supravaginal hysterectomy was done.

One may therefore feel reasonably dubious of the correctness of the generally accepted statement that cancer of the "cervical stump" represents from 2 to 4 per cent of cervical cancers.

TABLE I.—INCIDENCE OF CANCER OF THE "CERVICAL STUMP" IN RELATION TO CERVICAL CANCER

Author	Date	Cancer of cervix No.	Cancer of cervical stump—No.	Previous hysteromyomectomies—No.	Incidence of cancer of stump—Per cent
Behney, C. A.	1940	1117	24		2.15
		910	43	?	4.7
Faehndrich, J.	1934	860	12	8	1.28
Gagey, J.	1928	729	25		3.45
Healy, Wm., and Arneson, A. N.	1934	2600	67	39*	2.6
Hochman, S. S.	1926	263	7		1.14
Johnson, C. G. Tyrone, C. H.	1934	926	21	Not stated	2.2
Kretzschmar, N. R. and Gardiner, S.	1935	1022	18	11	1.7
Laborde, S. and Roques, A.	1928	379	11	6	2.9
Martzloff, K. H. Johns Hopkins Hospital, 1889 to 1917	1923	348	3	1	0.8
Martzloff, K. H. CollectedStatistics for The Johns Hopkins Hospital up to 1927	1934	512	9	6	1.7
Meigs, J. V.	1936	1218	23 occurred within 1 year of operation	12	1.9
			26 occurred after 1 year	15	2.1
			49	27	4.0
Scheffey, L. C.	1936	273	10	5	3.6

*58 per cent.

RELATIONSHIP OF CANCER IN THE RESIDUAL CERVICAL STUMP TO SUPRAVAGINAL ABDOMINAL HYSTERECTOMY

This is the most difficult and unsatisfactory phase of the entire problem, for if one is to obtain data comparable to other data on "cervical stump" cancer, it becomes necessary to know the outcome in the overwhelming majority of women who have had supravaginal hysterectomies for benign disease. The fact that cancer may develop in the residual cervix from 10 to 30 years after operation illustrates the necessity of tracing each patient until death if truly fundamental data are to be had.

Tesauro reported from Faure's clinic 10 (0.54 per cent) "cervical stump" cancers among 1,864 patients who had had supracervical hysterectomy. Operation was done for adnexal inflammation in 6 cases, myoma in 3, and ovarian tumor in 1.

Peham and Amreich encountered 8 instances of this complication among 1,253 patients who had had a supravaginal hysterectomy, an incidence of 0.638 per cent. In 1 of these the cancer was present at the time of the supravaginal hysterectomy, while in 1 it was recognized 7 months after operation and after 2 years in the third.

Faehndrich reported 2 instances (0.28 per cent) among 717 supravaginal hysterectomies. Scheffey reported 5 instances (0.9 per cent) among 554 patients. In 4 of these, however, the symptoms of vaginal bleeding developed in less than 1 year after operation and they may be assumed to have had unrecognized cancer of the cervix at the time of the first operation. Pearse reported 8 instances (1 per cent) among 810 patients, traced 5 or more years after supravaginal hysterectomy. Myomatous uteri had been previously removed in 410 of

these and 201 represented inflammatory disease without myomatous change. Each group revealed 4 instances of cancer in the residual cervical stump.

A compilation of the acceptable world literature by Faehndrich gives the incidence of cancer of the cervical stump following supracervical hysterectomy for benign disease as 0.39 per cent, while Albrecht puts the incidence at 0.32 per cent stating that he had encountered none in his material at Munich. Franz, according to Albrecht, traced 635 patients, who had had previous supravaginal hysterectomies, for a period of 18 years and encountered no cancer in the residual cervix. However, the shortcomings of all these compilations should be remembered.

The observation that supracervical hysterectomy may be done in the presence of unsuspected cervical cancer raises the question when a cervical stump cancer should be considered as having developed in a previously noncancerous cervix. Some writers, as previously noted, believe that cancer developing one year after operation should be considered satisfactory evidence that no cancer existed in the cervix at the time of the original supracervical hysterectomy. At the other extreme are those who believe that cancer developing in the residual cervical stump within 3 years after the original operation should be considered as probably existent at the time of the first operation. At first sight, the latter position may appear extreme and rigid. Granting that this concept is not always correct when applied to individual incidents, there is, however, much to support it.

A study of the peculiarly spaced time intervals that mark the manifestations of this complication, as noted by Herold, quickly arouses one's appreciation of this and suggests the pitfalls inherent in many statistics. The great bulk of cervical stump cancers may be divided into two major groups from the standpoint of the time of their occurrence after supravaginal hysterectomy (Fig. 1). One large group occurs during the first 3 years after operation and the other large group occurs after the 5th year following operation. Between the two there is a precipitate decline in the incidence of stump cancer. The regularity with which this occurs largely removes the observation from suspicion of mere coincidence or statistical error and, we believe, offers excellent reason for considering the great bulk of cervical stump cancers that manifest themselves during first 3 postoperative years as probably existent and unrecognized at time of original operation. A broad, rigid application of such a rule should result, in some inaccuracy. However, when applied to statistics it should prove an excellent, fair, and salutary yardstick.

Some of the statistics we have quoted earlier reveal that about 40 per cent or more of cancers of the cervical stump occur well within 3 years after operation. Confirmatory of this is a collective review by Faehndrich, of 299 instances of cervical stump cancer, in which the time of occurrence was noted as follows: 58, or 19.4 per cent, developed during the 1st year after operation; 68, or 22.8 per cent, developed between the 1st and 3rd year after operation; 42, or 14 per cent, developed between the 3rd and 5th year after operation; 131, or 43.8 per cent, developed 5 or more years after operation. Of this group in 73 cancer developed from 10 to 30 years after supracervical hysterectomy.

TOTAL VERSUS SUBTOTAL HYSTERECTOMY

In view of the fact that cancer may develop in the residual cervix after supravaginal hysterectomy for benign uterine disease, it has been proposed by some observers that performance of total hysterectomy as an inflexible routine would eliminate this complication. These observers also state that in competent hands the mortality from total hysterectomy is little if any more than that following subtotal hysterectomy and they advocate, therefore, without reservation, the total operation when hysterectomy is to be done.

On the other hand there are numerous opponents to the proposal that total hysterectomy be performed in every instance in which hysterectomy is to be done. This does not mean that panhysterectomy should never be employed for benign disease but that its use should be a matter of individualization and restricted to those instances in which there is an obviously pathological cervix, and the operation can be done without undue hazard. The basis for objection to total hysterectomy as a routine is that rigid adherence to the procedure leads to an increased operative mortality and morbidity; the operation is ordinarily inadequate to cure patients who have an established but unsuspected cervical cancer and, the hazard of subsequent cancerous change in a previously noncancerous cervix is not as great as the increased mortality inherent in the total operation. Finally, the objection has been raised that the total operation is no guarantee that cancer may not develop later in the vaginal vault.

The relative mortality of these two operative procedures is difficult to compare. It is obvious that when exposure is difficult or the patient is or has become a poor surgical risk because of hemorrhage or some other acute contingency, the supravaginal operation is the only procedure possible or permissible. Staunch advocates of the routine use

of total operation, such as Polak and Culbertson, have receded from their positions, because of an unsatisfactory experience. Others have gradually adopted the procedure as a routine, some recognizing as do Weir, Wertheims, Weibel, and others, that this entails greater danger of injury to bladder and ureters. Weir reports a morbidity of 2.4 per cent arising from bladder and ureteral injury alone, while Fullerton and Faulkner report an incidence of 1.4 per cent. None of these complications occurred in their group of supravaginal hysterectomies. However, some of the zealous advocates of routine total hysterectomy deny that it possesses any appreciable increased hazard and apparently do not recognize the existence of situations in which the procedure is well nigh impossible. The overwhelming weight of competent gynecological opinion, however, is of the opposite view and considers the total operation as one of election to be done only when the indication exists and the operation does not involve undue hazard or frank foolhardiness.

The question of morbidity after these operations may be mentioned briefly. During the developmental phase of the operation for hysterectomy one of the difficult problems facing the surgeons of that time was the development of a technique suitable to manage the cervical stump after supracervical hysterectomy. Hemorrhage and particularly infectious complications on the proximal side of the stump were factors that prompted some surgeons to forsake the supravaginal procedure for the more complicated total hysterectomy. These complications arising from the cervical stump when mass ligatures were used with subsequent ischemic necrosis are no longer important when acceptable techniques now available are employed. If we omit injury to bladder and ureters, complications particularly inherent in total hysterectomy, it is difficult to determine from the literature that other complications, such as, fever, thrombosis, and embolism are less common after total hysterectomy. Postoperative morbidity as indicated by fever of unexplained origin is, as well as we can ascertain, probably no more common after the subtotal than after the total operation.

I have never seen a fatality in which autopsy showed the cervical stump to be the cause of postoperative death in a manner that does not occur following the total operation. Intestinal obstruction is possible after either operation, but prolapse of the intestine into the vagina is possible only after the total operation.

Reference to Table II shows among other things the wide variations that may occur in surgical fatalities from the same type of operation.

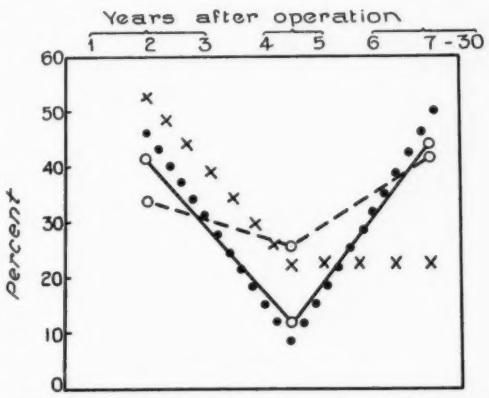


Fig. 1. Graph illustrating peculiar curve of incidence of cancer in residual cervix after supracervical hysterectomy. o—o Faehndrich collective review; o---o Faehndrich personal series; . . ., Waldeyer series; x x x, Meigs series.

Probably not only the skill but judgment of the individual surgeon and indications he considers adequate to justify operation play a large part in this.

It is impossible to know from these figures which ones represent hysterectomies that were done on normal sized uteri for mild pelvic inflammatory disease or for small myomas which may offer to some conservative gynecologists dubious indications for operation. These observations apply particularly to total hysterectomies. Also, it is difficult to compare figures, such as Waldeyer's on uncomplicated myomas, who failed to report mortality on his entire series, with those of Kelly, Cullen, TeLinde, Miller and Prejean and others, which represent consecutive "run of the mill" material from clinics where much of the material represents advanced and complicated pathological lesions. When comparative mortality statistics by a single author reveal a higher mortality for the supravaginal operation than for the total operation, it is difficult to escape the impression that the supravaginal operation, as in Miller's and Prejean's series, was done in the difficult and complicated cases. If both types of operation were done on comparable types of cases there is little doubt that mortality and morbidity as suggested by Aldridge would be uniformly higher following total hysterectomy. The figures of TeLinde represent the nearly ideal results that may be obtained by a competent group of surgeons when the total operation is used as an elective procedure and not forced as a fixed routine. Finally the collected statistics of Albrecht and of Doederlein show the wide fluctuation that can occur in such compilations.

TABLE II.—COMPARISON OF MORTALITY RATES OF TOTAL AND SUPRAVAGINAL HYSTERECTOMY

Author	Yr.	Number of operations (all abdominal)	Mortality per cent	
			Total hysterectomy %	Supravaginal hysterectomy %
Amreich, I.	1925	For myomas	6	1.6
Danforth, W. C.	1934	350		0.28
		744		0.8
	1941	150	0.66	
Doederlein	1924	90	5.5	
Dupertuis, S. M. and Zollinger, R. (Peter Bent Brigham Hospital)	1938	514		1.7
		118	2.8	
Faehndrich, J.	1934	235 uncomplicated myomas		1.5
			4.2	
		707		4.5
		304 all benign disease	6.25	
Franz, K. (quoted by Waldeyer)	1928	635		1.6
		302	3.7	
Fullerton, W. D. and Faulkner, R. L.	1930	636		4.2*
		1078	4.1	
Kelly-Cullen (Supravaginal hysteromyomectomies of all types.) (The Johns Hopkins Hospital)	1909	901 (years 1889–1906)		5.5
		192 (years 1906–1909)		1+
Kroenig	1924	258	5.8	
Masson, J. C.	1940	766		0.9
		1776	1.2	
Meigs, J. V. (Massachusetts General Hospital)	1934	1771		2.9
		224	4.4	
Miller, H. E. and Prejean, O.	1941	255		2.75
		374	1.33	
Pearse, R. L. (Free Hospital for Women, Brookline, Mass.)	1934	1900		1.7
Read, C. D. and Bell, A. C.	1933	1607		2+
		573	3.3	
Rinaman, J. C. and Sellers, T. B.	1935	179		2.8
		62	9.6	
Siddal, R. S. and Mack, H. C.	1935	1141		2.6
		235	6.4	
TeLinde, R. W. (Personal communication) (All types of benign disease) (The Johns Hopkins Hospital)	1942	663		0.15
		152	0	

*This figure is generally quoted as 4.4% but this we cannot verify.

TABLE II.—Continued

Author	Yr.	Number of operations (all abdominal)	Mortality per cent	
			Total hysterectomy %	Supravaginal hysterectomy %
Waldeyer, L. (Report concerns 824 total hysterectomies, but does not give mortality figures for all of these.)	1934	320 uncomplicated myomatous uteri	2.2	
Weibel (quoted by Waldeyer)				4.25
			3.5	
Weir, W.	1931	663	3.1	
	1938	531	2.6	
Albrecht, H. (collected statistics)	1928	4575		1.9
		2305	3.6	
Doederlein (Doederlein-Kroenig collected statistics.)	1924	2642		4.9
		2156	4.9	

The fact that cancer of the vaginal vault, as reported by Meigs, Tyler, and others, occurs occasionally after panhysterectomy, has been used as an argument that panhysterectomy is not the panacea, as claimed by some, against future development of cancer at the former site of cervix.

This argument possesses undoubted validity, but it is not as conclusive as it sounds when one reflects that cancer of the vagina may occur in the presence or absence of the cervix uteri. The 13 cases ascribed to Tyler represent instances reported in the French literature, and essential details are lacking. Also when cancer occurs in the vault after a previous panhysterectomy one can not be certain, unless one has had an opportunity to study carefully the original operative specimen, that the entire cervix had been removed, or that it may not have harbored an unrecognized cancer.

The value of total hysterectomy for cancer prophylaxis is granted. However, as we view the situation, the important consideration in deciding to do an abdominal panhysterectomy is not the presence of a myomatous uterus, or the obvious desirability of removing simultaneously a symptom producing cervix, but the ability to do the operation without additional risk to the patient. The presence of benign disease of the cervix and the fear of subsequent cancerous change, is, we believe, no special indication for panhysterectomy if this procedure entails an increased operative hazard. The so called "angry, red appearance" of a bleeding circumostial vermilion zone offers real fuel to the suspicion that the fire of cancer is

near at hand. The statement is therefore frequently encountered that an "eroded" inflamed cervix offers special danger of subsequent development of cancer. There is, however, little if any factual information to support this constantly repeated assumption. All of the truly early *bona fide* cancers we have seen so far have occurred on relatively normal appearing cervices. It is, I believe, extremely doubtful that one can foretell from the appearance of a cervix involved in a benign inflammatory process whether it is more likely to develop cancer than a normal appearing cervix. Nulliparity is no guarantee against subsequent cancer development. Therefore, the admonition that when hysterectomy is indicated for benign uterine disease, a diseased cervix is a strict indication for panhysterectomy, because it is particularly prone to develop cancer if left *in situ*, becomes largely a matter of individual opinion, unsupported, as far as we can ascertain at this time, by acceptable proof.

SUMMARY

The statistical data on which are based some of the arguments regarding this controversial subject, are in some respects unsatisfactory and inconclusive. It appears, however, that the incidence of cancer in the residual cervix after supravaginal hysterectomy is less than 1 per cent. Total hysterectomy, except when used as an elective procedure by well qualified individuals has a mortality rate definitely in excess of the supravaginal operation. The difference in mortality between the two operative procedures is evidently in excess of the incidence of *bona fide* cancer which may develop in the residual cervix *after* supravaginal hysterectomy. There is good reason, therefore, to doubt the wisdom or factual basis for advocating panhysterectomy as a fixed *routine* when hysterectomy is to be done for benign uterine disease in order to avoid the possibility of subsequent cervical cancer.

There can be little question of the desirability of panhysterectomy as prophylaxis against subsequent cancerous change in a residual cervical stump when the operation is used as an elective procedure without additional hazard to the patient.

There is good reason to believe that as a general rule cancer manifesting itself in a residual cervix within 3 years after supravaginal hysterectomy for benign disease was present but unsuspected at the time of the original operation.

The admonition that benign inflammatory disease of the cervix offers particular predisposition to later cancerous change is open to question.

REFERENCES

1. Ahlquist, R. E. West. J. Surg., 1942, 50: 109.
2. Albrecht, H. Klinik des Myoma Uteri. Halban-Seitz, Biologie und Pathologie des Weibes. Berlin: Urban & Schwarzenberg 1928, 4: 387, 472.
3. Aldridge, A. H. Am. J. Obst., 1939, 37: 985.
4. Amreich, I. Zschr. Geburtsh. Gyn., 1925, 88: 68.
5. Behney, C. Am. J. Obst., 1940, 40: 780.
6. Bowers, D. D. Am. J. Obst., 1940, 39: 830.
7. Culbertson, C. Personal communication, 1932.
8. Danforth, W. C. Am. J. Obst., 1941, 42: 587.
9. Doederlein-Kroenig. Operative Gynäkologie. Leipzig: Georg Thieme, 1924.
10. Dupertuis, S. M., and Zollinger, R. Surg. Gyn. Obst., 1938, 67: 689.
11. Faehndrich, J. Zschr. Geburtsh. Gyn., 1934, 109: 382.
12. Fullerton, W. D., and Faulkner, R. L. J. Am. M. Ass., 1930, 95: 1563–1567.
13. Gagey, J. Discussion of paper by Laborde and Roques (19).
14. Graff, E. v. Tr. Am. Ass. Obst. Gyn., Abd. Surg., 1931, 44: 167.
15. Healy, W. P., and Arneson, A. N. Am. J. Obst., 1935, 29: 370.
16. Herold, K. Zbl. Gyn., 1933, 57: 108.
17. Johnson, C. G., and Tyrone, C. H. Surg. Gyn. Obst., 1934, 58: 113.
18. Kretzschmar, N. R., and Gardiner, S. Am. J. Obst., 1935, 29: 168.
19. Laborde, S., and Roques, A. Bull. Soc. obst. gyn. Paris, 1928, 17: 123.
20. Martzloff, K. H. Diseases of the Cervix Uteri. Lewis' Practice of Surgery, Vol. X. Hagerstown, Md.: W. F. Prior Company, Inc., 1940.
21. Masson, J. C. Am. J. Surg., 1940, 48: 255.
22. Meigs, J. V. Tumors of the Female Pelvic Organs. New York: The Macmillan Co., 1934.
23. Idem. Am. J. Obst., 1936, 31: 358.
24. Miller, H. E., and Prejean, O. Am. J. Obst., 1941, 42: 580.
25. Pearse, R. L. Surg. Gyn. Obst., 1934, 58: 845.
26. Read, C. D., Bell, A. C. J. Obst. Gyn., Brit. Empire, 1933, 40: 749–767.
27. Richardson, E. H. Am. J. Obst., 1935, 30: 237.
28. Rinaman, J. C. and Sellers, T. B. Am. J. Surg., 1935, 27: 282.
29. Siddall, R. S., and Mack, H. C. Surg. Gyn. Obst., 1935, 60: 102.
30. TeLinde, R. W. Personal communication, 1942.
31. Tesauro, G. Rev. mens. Gyn. Obst., 1928, 18: 3. (Abstr. Zbl. Gyn., 1929, 53: 2307.)
32.[1] Tyler, G. T. South. M. J., 1915, 8: 583.
33. Waldeyer, L. Zbl. Gyn., 1934, 58: 35.
34. Weibel, W. Die gynäkologische Operationstechnik der Schule Ernst Wertheims. Berlin: Julius Springer, 1923.
35. Weir, Wm. H. Ohio M. J., 1938, 34: 270.

[1]The 13 cases of cancer in the vaginal vault attributed to Tyler are reported in the discussion of a paper by M. Chaput. Bull. Soc. chir. Paris, 1910, 36: 610.

REPAIR OF THE INCONTINENT SPHINCTER ANI

PAUL C. BLAISDELL, M.D., F.A.C.S., Pasadena, California

IN previous papers some of the general facts and specific problems associated with injuries to the sphincter ani muscle and its surgical repair have been discussed (1, 2, 3). Particular attention was called to material deficiencies of what had been commonly regarded as a satisfactory operation for the relief of incontinence, and a report was given of our experience with an operation which gave promise of certain definite advantages, amply established in our hands. Since that time further personal experience with this latter operation together with reports from other surgeons has but strengthened our confidence in it. We therefore turn now to a different phase of the problem.

First, however, in evaluating the importance of this general topic let us emphasize that much surgery of greater technical magnitude may actually represent far less in ultimate import. For let it not be lost sight of that a successful operation in this field does not just restore the function of a small muscle. In the truest sense it rehabilitates an otherwise useless, ruined life to grateful existence. Only one who has dealt with these patients appreciates the full meaning of this truth.

Intelligent discussion of reparative surgery of the incontinent sphincter ani must be based on the recognition of three rather clear cut groups of cases. The failure to distinguish between such divisions as presenting quite different problems has heretofore rather clouded the issues. For example one hears comparative approbation of different operations when actually their design served totally different purposes. For more orderly consideration therefore we suggest a strict anatomical grouping on the basis of relative amounts of intact functioning muscle remaining and available to the surgeon as a foundation for repair.

The great majority of cases even with complete incontinence involve comparatively small segmental deficiencies of the muscle. This is readily understood when one reviews the causes involved (2). It is this major class and only this class for which the classic plastic repair is dubiously useful, and it is this class which we discussed in our first paper and for which we proposed the simpler and more satisfactory operation therein described.

From the Department of Proctology, College Medical Evangelists, and the Rectal Service, Los Angeles County General Hospital

The other anatomical extreme fortunately involves numerically the fewest cases and is comprised of those instances in which the muscle is either congenitally absent or totally destroyed for all practical purposes by exceptional circumstances. In this relatively small class there is an entirely different problem of constructing a complete new sphincter from totally extraneous voluntary muscle tissue, usually the glutei.

The middle ground is comprised of those cases in which a large segment, say up to approximately a half of the sphincter, has been destroyed. It would seem hopeless on the one hand to make reparative use of any smaller amount than this of remaining muscle and yet this much muscle or any more seemed too much to just cast aside. On the other hand, from the surgical standpoint there is the distinguishing problem of excessive postoperative tension were the usual plastic repair attempted. While our own reefing operation, so successful in the first group, is obviously inapplicable when a large segment of muscle has been destroyed; because instead of converting the muscle into a functioning circle of smaller circumference it would tend simply to flatten out the effective portion remaining. Heretofore it has been the characteristic history of cases in this intermediate group to be subjected to one or more totally unsuccessful attempts at suturing the dissected ends of remaining muscle and then to be relegated to candidacy for the complicated operations of foreign muscle transplantation which constitute the only hope of the extreme group mentioned. We therefore sought to devise some satisfactory technique to take advantage of an appreciable amount of good muscle tissue and rescue such cases from the class of total absence. This is the topic of the present paper.

Considerable difficulty from several sources might be surmised in differentiating clinically the three groups and accurately evaluating the varying amount of functioning muscle remaining. In the first place the surgeon who is finally called upon to repair the damage is not usually the one who has followed the case to that point and thus had the benefit to orientation of participating in previous dissections. But even more importance attaches to the fact that the degrees of physiological and anatomical damage have little parallel relationship; while incontinence of gas alone can only

Fig. 1.

Fig. 2.

Fig. 3.

Fig. 1. Photograph of case reported, illustrative of incontinent anus with extensive destruction of the sphincter muscle and with scarring of previous unsuccessful attempts at repair. In this case only the anterior half of the sphincter remains, as indicated by overlying puckered skin. Anal opening widely patulous.

Fig. 2. Photograph of same case 2 weeks after first stage operation, with incision completed for second stage operation. Anal opening has been considerably diminished by the transplantation of muscle-bearing flaps, with the sphincter now surrounding approximately ¾ of the anal circumference.

Fig. 3. Photograph taken 10 days following second stage operation. One stay suture only remains. Anal opening is completely closed and the sphincter now completely encircles it.

occur from minor injury, yet on the other hand complete incontinence may supervene from very little more, or even the same amount elsewhere on the circumference. All three grades of incontinence (to passage of gas, liquid, and solid stool) may occur in our first anatomical group. As a matter of fact, though, the determination of anatomical injury is usually rather easy, because the skin overlying intact muscle is thrown into folds by contraction of the latter, while the skin overlying any defect is not so affected and hence tends to be smoothed out. Figure 1 illustrates this quite dependable finding. In cases of relatively minor injury a scarred and sometimes slightly depressed notch marks the area of previous injury and separation of the muscle ends.

In devising a possible new or improved type of operation for the group under discussion we naturally turned first to an analysis of the causes of failure with the usual suture of the muscle ends freed by dissection. With such a large defect to be bridged the outstanding shortcoming was unquestionably the extreme tension to which the sutured muscle ends were necessarily subjected. This hazard is precarious enough when only small deficiencies exist as indicated by our previously reported statistics. In the case herewith reported, exemplifying the group with much more serious defect, half of the muscle circumference had been destroyed. Under such circumstances, and in accordance with the physical laws of elasticity, even if the whole remaining half could be completely mobilized and utilized the tension would be enormously increased. Considerations of tissue viability, which limits the amounts of muscle tissue which can be safely freed by dissection, further restrict the potentialities.

Again, dissecting out the frayed muscle ends is in itself likely to be a difficult and protracted procedure even to one familiar with the regional anatomy, for the ends of the mutilated muscle are lost in a maze of dense scar. Furthermore, and at best, an appreciable amount of probably functioning tissue is sacrificed in reaching undamaged fibers in which clear-cut delineation of muscle belly is possible. Hence in this group in which the amount of remaining muscle is already of critical concern, this stage, difficult enough in cases of minor damage, looms as an even greater obstacle in the problem of bridging large defects. It must be admitted also that muscle tissue does not lend itself to suture under tension, being particularly susceptible to tearing when the sutures pull in the direction of the fibers, as here. And the vigorous trauma of protracted dissection certainly adds nothing to their strength.

A further obstacle interposed is that of inevitable wound infection with varying degree of

Fig. 4. First stage of operation, showing lines of incisions, commenced on left. This gives a tongue-shaped flap of muscle-bearing tissue on each side with the tip A to be transplanted and fixed at A^1. It is important to pattern the incision and to carry it deep enough to be certain of mobilizing muscle. We did not undermine flaps but this feature could be considered with propriety.

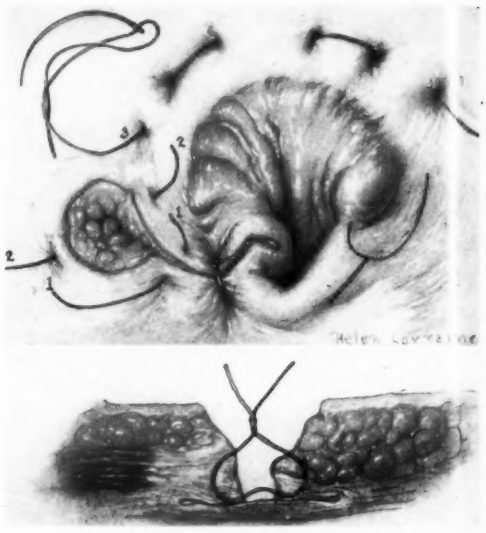


Fig. 5. Deep buried suture of fine wire as shown in inset, has approximated points A and A^1 of Figure 4. (Wire is tied however, not twisted as shown, and cut short—just as gut suture.) One stay suture has been tied—this is not the wire suture shown in inset; two others, *1*, *2*, have been placed. These were carried deep to operative wound for considerations of infection. Pursestring suture is shown started around operative field, though actually this is not carried out until completion of both sides.

subsequent disruption. This is of course not unique to this group and we mentioned it in our previous consideration of minor injuries. But with increase of tension this factor assumes increased significance. Fundamentally, any suture of an anal wound is a compromise with ideal proctological practice. Our efforts in combating infection represent nothing new but simply a combination of promising suggestions.

All factors considered it is little wonder that this operation gave little or no chance of success in connection with more serious injuries and extended defects.

We therefore planned and herewith report a procedure to obviate or minimize these shortcomings. And for it we suggest the following advantages:

1. By a *two stage operation* involving *gradual transplantation* of the muscle ends in *steps* postoperative wound tension is reduced to proportions compatible with reasonable chance of success. Indeed through the use of our operation, tension is entirely under control of the operator and could even be reduced almost *ad libidum* by additional operative stages. We emphasize that such control in itself may mark the whole difference between success and failure in the rehabilitation of the patient. In addition, and as a more minor expedient, a temporary purse string of tension suture was introduced immediately after operation at each stage completely to surround the operative site for the same purpose, controlling tension.

2. Instead of dissecting out and suturing the muscle ends themselves, we utilized as a second major principle almost instantaneously shaped and transplanted flaps of *muscle bearing* tissue. This provision in itself marks a radical departure in concept and procedure. We reasoned that material contribution would thereby be simultaneously made not only to tremendous simplification of the operation but also to strength of the suture line, and to preservation of every fiber of remaining muscle. Such additional strength would be derived from two sources, viz., availability of tissue for suture better suited to withstand tension, and increased amount of such tissue available for suturing in distinction to the more limited muscle belly. We could not see anything to be lost from a theoretical standpoint. For muscle has to be surrounded ultimately by other tissue, either new or old, and it is a rather universal law that, other things equal, living cells lose rather than gain by transplantation to new soil. And here, under the old regimen, any subsequent new soil would be preponderantly scar tissue anyway—the same kind of tissue removed at such arduous pains. Of importance also is the fact that

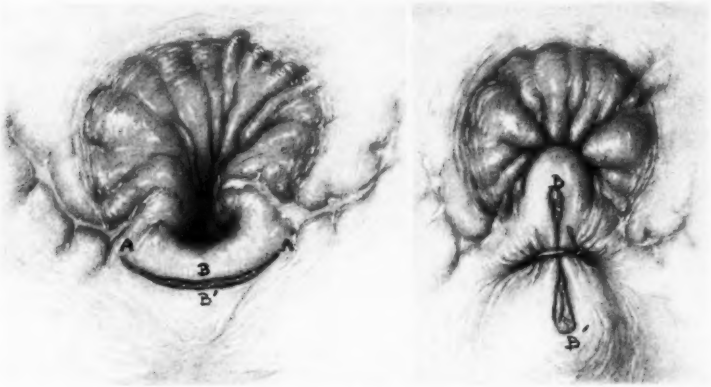


Fig. 6. Second stage of operation. a, Incision commenced, again to be carried to greatest depth of sphincter muscle. b, Points A and A^1 have been approximated by deep wire suture as shown in Figure 5 inset and one stay suture has also been tied—and too tightly as shown here. Notice how this type of operation closes anal opening without leaving a crevice wound directly contaminable from inside of anus. Removal of a "piece of pie" shaped segment, on the other hand, could accomplish the same approximation of the muscle ends but would leave a wound the depths of which would be in direct communication with the anal canal; gross contamination would in such case inevitably result either in breaking down of the wound, or, if the latter healed, in a fistulous tract. This simple second stage procedure here illustrated might of itself supplant admirably the commonly used and more formidable plastic procedure (2) for minor deficiencies.

with our operation there is involved no sacrifice of functioning muscle tissue such as is inevitable in dissecting out the frayed ends of the sphincter.

While this transplantation of a muscle bearing flap was designed for the operation under discussion we see no reason why it is not applicable to great advantage in connection with the usual plastic repair of minor injuries. Or again it might be used for additive effect when our reefing operation fails to give complete satisfaction, or when inapplicable.

3. The operation devised is extremely simple. We early resolved that simplicity was so essential from every standpoint that we would discard anything of other attribute. But our success exceeded all we dared hope and we now are able to substitute a simple operation involving the work of actually but a few minutes for a long tedious undertaking, and with not only equal but far surpassing effectiveness. There are no superficial flaps to be raised for purposes of exposure, then to be closed; and the laborious and precarious dissection of muscle ends frayed and scattered in scar tissue is eliminated in even semblance. Such simplicity contributes directly in obvious manner and also indirectly in intangible but important respects such as diminishing trauma and the incidence and devastation of infection.

4. The incidence and effects of infection were opposed by the use of single buried stay sutures of alloy steel wire at critical points, the introduction of a sulfonamide drug directly into the wound, and the exclusive use of circumferential incisions which prevent direct leakage into the wound after operation from within the anal canal; these in addition to well recognized preoperative and postoperative precautions. We have also mentioned the elimination of inordinate operative trauma as a contributing factor.

The case reported herewith as illustrative of our technique is valuable to our purpose because it is tragically typical in deplorable history and distressing surgical sequence. But most important it served as a rather critical control in evaluating the merits of our procedure because the patient had been previously subjected with total failure to repeated reparative operations which sought to make use of the sphincter residuum, and to transplantation operations which attempted employment of extraneous muscle. These operations had been performed by capable proctologists in whose competence we have confidence. And while no one can say that it is either warranted or unwarranted to assume that the utmost possibilities inherent in these operations had been exhausted, it is obvious that they had had fair and repeated

trial by skill available to comparatively few patients. In contrast, our own result, accomplished after all else had failed, was eminently satisfactory to the patient, and, by virtue of an operation readily available in performance to a far larger body of surgeons, a correspondingly widened patient group could be benefited. Furthermore it is pointed out that every attempt at repair of these cases makes succeeding attempts both more difficult and precarious. This should mean even more hopeful anticipation should our suggestions be carried out for there would be the added benefit of primary attempt at restoration.

The patient whose condition is shown in the accompanying photographs gave the usual history of having been subjected over a period of years to numerous operations for a fistula-in-ano. The kindest thing one may say of the result is that it represents the fruit of some surgeon's or surgeons' total incompetence in this field. It left the patient so completely incontinent that with a desire for defecation he could not reach an adjoining bath room in time. And with all this the fistula was still uncured! With the hopelessness and despair of years he had become a derelict, utterly useless to society and devoid of any semblance of self respect or interest.

He first entered the proctological service of the Los Angeles County Hospital in 1935 at which time the fistula was easily eradicated. During the succeeding several years repeated attempts were made to improve his condition of complete incontinence. Both reparative and transplantation procedures were attempted with no improvement whatsoever.

The patient was first admitted to my service in November, 1941. Figure 1 is a photograph taken at this time and shows the widely patulous anal opening. Palpation revealed the power of voluntary contraction but without appreciable effect in closing the sphincter. At this time we performed the first stage of the operation. Incisions were made on both sides of the anus, being carried to the level of the depth of the sphincter muscle. No attempt was made to undermine the muscle bearing flaps and no tissue whatsoever was removed. The two opposing ends of the incision were simply drawn together, held first by a single deeply buried suture of alloy wire. Thus the muscle bearing flap was transplanted to its new location. The resulting opposed edges on either side of the flaps were fitted together and held loosely with interrupted tension suture layed deeply to the wounds involved. After completion of both sides a pursestring of tension suture was introduced surrounding the whole anus and pulled and tied tightly, thereby removing the last vestige of tension on the wounds. Postoperative care followed well recognized lines.

Figure 2 is a photograph of the result two weeks after the first stage. The patient could now inhibit bowel movement for 40 minutes after the urge and for the first time in years he had the feeling of effective power in the sphincter ani.

The same simple principles were followed in the second stage which, however, was even simpler in point of actual technique. It consisted simply of a curving incision to the depth of the sphincter muscle base with apposition of the ends by suture. Again we used a buried suture of alloy steel as an anchor and other aids previously employed. Figure 3 is a photograph of the condition 10 days after the second stage and shows the transition to a firmly closed canal.

It is of course difficult to measure the results of any operation for incontinence in specific and accurate terms, and it is not usual to err on the side of under enthusiasm. It would be preposterous to say this man's sphincter is as good as it ever was. On the other hand his abundant gratitude bespeaks a worthwhile result. We should prefer, however, to rest on the reasonableness of our approach and upon its trial by those to whom it may appeal.

SUMMARY

Plastic repair of large defects of the sphincter ani muscle is discussed. A very simple yet effective reparative operation is described involving the *gradual transplantation* in *multiple stages* of *muscle bearing* flaps. The chief advantages over the one stage operation in common use are the complete elimination of disastrous postoperative tension and the tedious and mutilating dissection of frayed muscle ends. Also it restricts the necessity of complicated transplantation of extraneous muscle.

REFERENCES

1. Blaisdell, Paul C. J. Am. M. Ass., 1939, 112: 614.
2. Idem. Surg. Gyn. Obst., 1940, 70: 692.
3. Ibid., 1937, 65: 672.
4. Smiley, Kenneth E. Tr. Am. Proct. Soc., 1940, 191.

COMPLICATIONS OF INFEROANTERIOR (AXILLARY) DISLOCATION OF THE SHOULDER JOINT AS DEMONSTRATED BY ROENTGENOGRAMS

MAURICE D. SACHS, M.D., and HAROLD A. HILL, M.D.,
San Francisco, California

THE grooved or "typical" defect in the humeral head has been described by early anatomists and surgeons. In 1861 Flower reported 41 shoulder specimens which he had collected from pathological museums in London, including cases with fractures of the greater tuberosity, grooved defects in the posterior head of the humerus, and dislocated bicipital tendons on the greater tuberosity. In 1880, Eve reported the first proved case of grooved defect. His patient died 12 hours after a train accident in which his shoulder had been dislocated. Postmortem examination of the humerus revealed a groove of recent origin in the lateroposterior portion of the head. Many additional cases were reported by Kuster (1882), Loebker (1887), Schuller (1890), Stoeffel (1895), Francke (1898), and Wendel (1903). Since humeral head resections have been replaced by the conservative methods of Nicola and Bankhard, the operating surgeon has had very little opportunity to observe the defect. Roentgenologists, however, by means

From the Station Hospital, Camp Callan, California, and the Department of Medicine (Radiology), Stanford University Medical School.

Read before California State Medical Society, at Del Monte, California, May 5–8, 1941, and the Western Orthopedic Association, San Diego, California, November 14–15, 1941.

Dr. Sachs, Captain, M. C., U. S. A.; Dr. Hill, Lieutenant Commander, M. C., U. S. N.

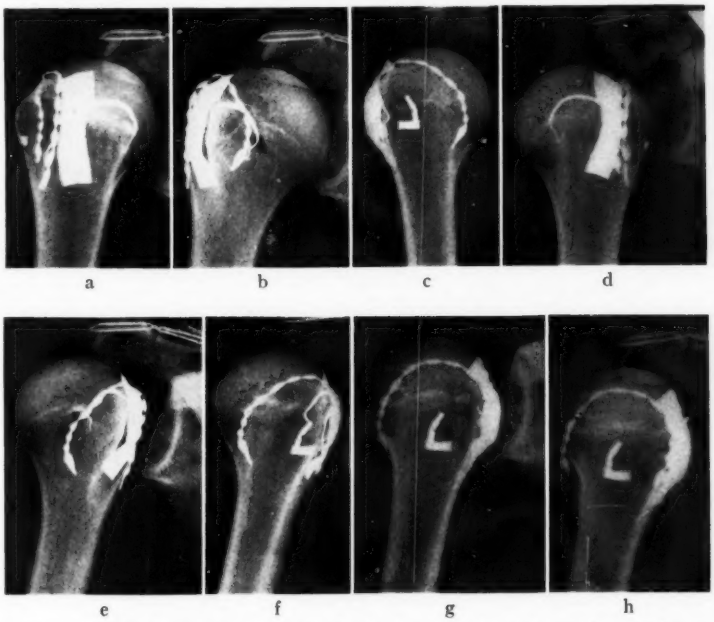


Fig. 1. Skeletal roentgen studies beginning in external rotation and gradually externally rotating humerus until complete circle is rotated. a, b, Reveal greater tuberosity in profile view. f, g, h, Reveal the lateroposterior head of the humerus in profile view.

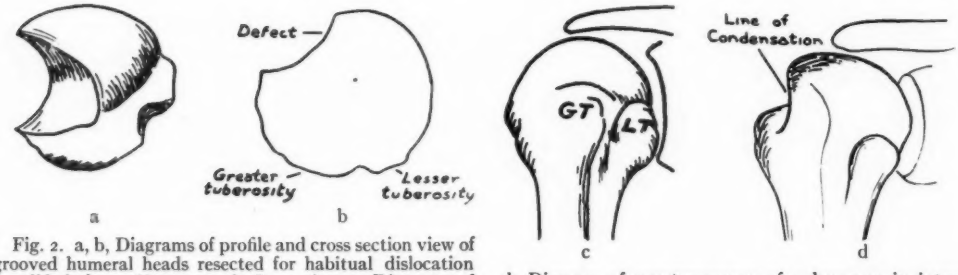


Fig. 2. a, b, Diagrams of profile and cross section view of grooved humeral heads resected for habitual dislocation (modified from Kuster and Cramer). c, Diagram of roentgenogram of a normal humerus in internal rotation. d, Diagram of roentgenogram of a humerus in internal rotation, showing defect.

of fluoroscopy and roentgenograms have been able to diagnose this phenomenon. Perthes (1906) described 2 cases, Schultze (1914) 5 cases, Pilz (1925) 15. Hermodsson reported that it occurred at the initial examination and need not change appreciably in subsequent dislocations. One of his patients, following a fall, revealed a normal roentgenogram of shoulder; but several months later after dislocation of the same humerus immediate roentgenogram showed a typical defect.

Bone anatomy of the shoulder joint. Thorough knowledge of the topographic anatomy of the shoulder joint and mechanics involved in dislocation is essential to a better understanding of roentgenographic complications which may be the result of inferoanterior or so called subcoracoid dislocations. To simplify topographical roentgenographic anatomy of the shoulder joint, wires are placed about both tuberosities, a smooth wire about the lesser and a twisted wire about the greater (Fig. 1). The letter "L" is placed on the prominent portion of the greater tuberosity and lead foil in the bicipital groove. Roentgenograms are taken with the humerus gradually rotated externally until a 360 degree circle is completed. The first film is taken with the hand in supina-

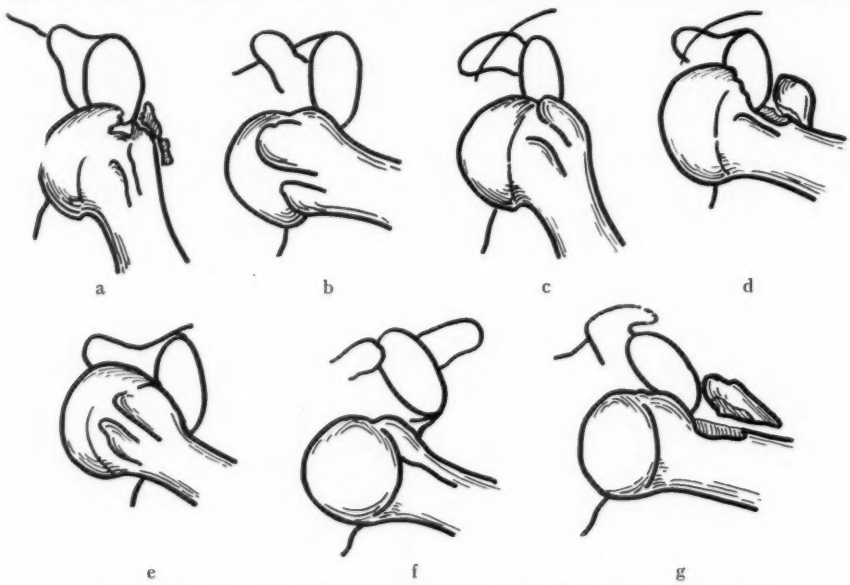


Fig. 3. Diagrammatic sketches from roentgenogram studies, revealing various steps in so called inferoanterior or subcoracoid dislocation. a, Subglenoid external rotation; b, inferoanterior internal rotation-axillary; c, inferoanterior external rotation-axillary; d, subcoracoid external rotation; e, subcoracoid internal rotation; f, inferoanterior external rotation-axillary; g, inferoanterior external rotation-axillary.

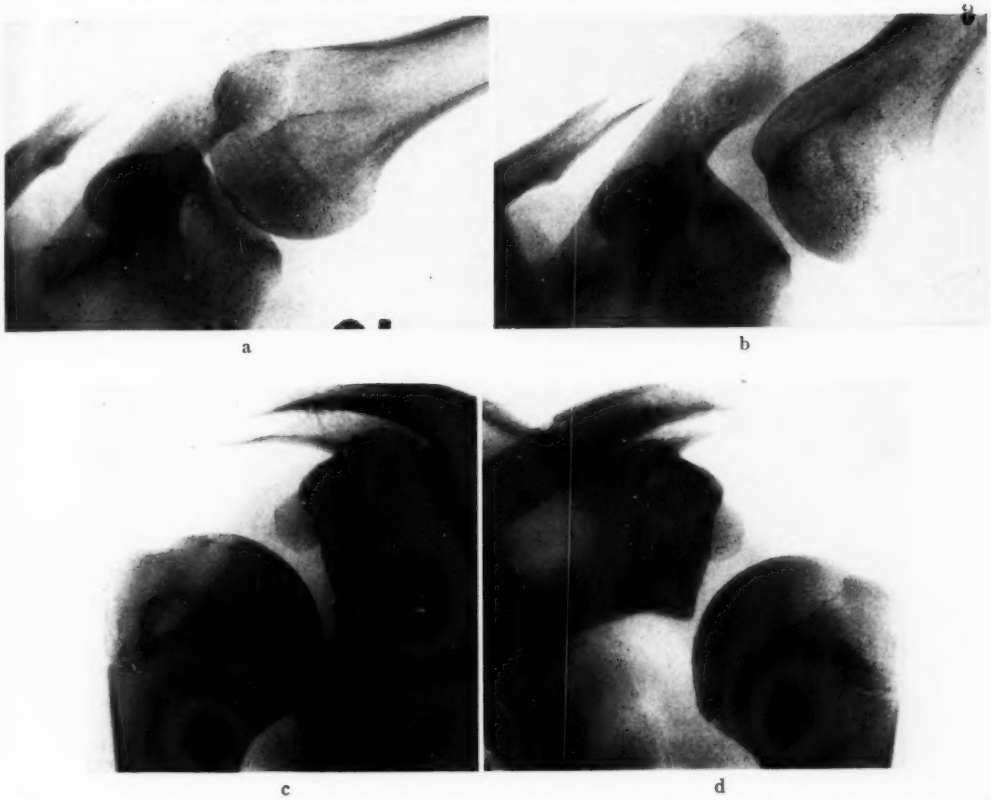


Fig. 4. a, Case 1. Left humerus in abduction is within normal limits. b, Same humerus with voluntary movement on the part of the patient reveals a subglenoid subluxation. c, Case 2. Right humerus in forced extension reveals a subglenoid subluxation. Note the site of the Nicola operation. d, Left humerus in forced extension reveals a similar subglenoid subluxation (apparently there is an anomalous maldevelopment of the glenoid articulating rims).

tion, that is, external rotation, and only a small portion of the greater tuberosity is seen in profile view (Fig. 1, a). The greater portion of the humeral head consists of the lesser tuberosity and the bicipital groove. However, in internal rotation the bicipital groove overlaps the glenoid and the lesser tuberosity is rotated inwardly and posteriorly (Fig. 1, f, g, h). The prominent portion of the humeral head consists of the entire greater tuberosity, posterior portion of the anatomical neck, and a part of the lateroposterior humeral head. This latter view is especially important inasmuch as this area is very frequently fractured during the course of dislocation. If a film were taken in external rotation, this region would not be visualized and the fracture would be missed (5). For this reason, routine films of the shoulder should be taken at least in external and internal rotation, thus enabling an x-ray demonstration of at least three-fourths of humeral head.

Complications of inferoanterior or subcoracoid dislocations are: so called compression defect in the lateroposterior head of the humerus (3, 4, 5, 10), fracture of the greater tuberosity and fracture of the inferoanterior lip of the glenoid. Somewhat rarer are the findings of cystic changes (3, 4, 5) in the head of the humerus and in the region of the bicipital groove. These complications will be considered and illustrated later.

Mechanics of inferoanterior dislocations. The mechanism of the so called inferoanterior (axillary) or subcoracoid dislocation has been described by numerous authors (2, 3, 6, 8, 9, 11, 12). Few of these theories are in agreement and none are entirely acceptable to us (the authors' original conception was not entirely correct). It

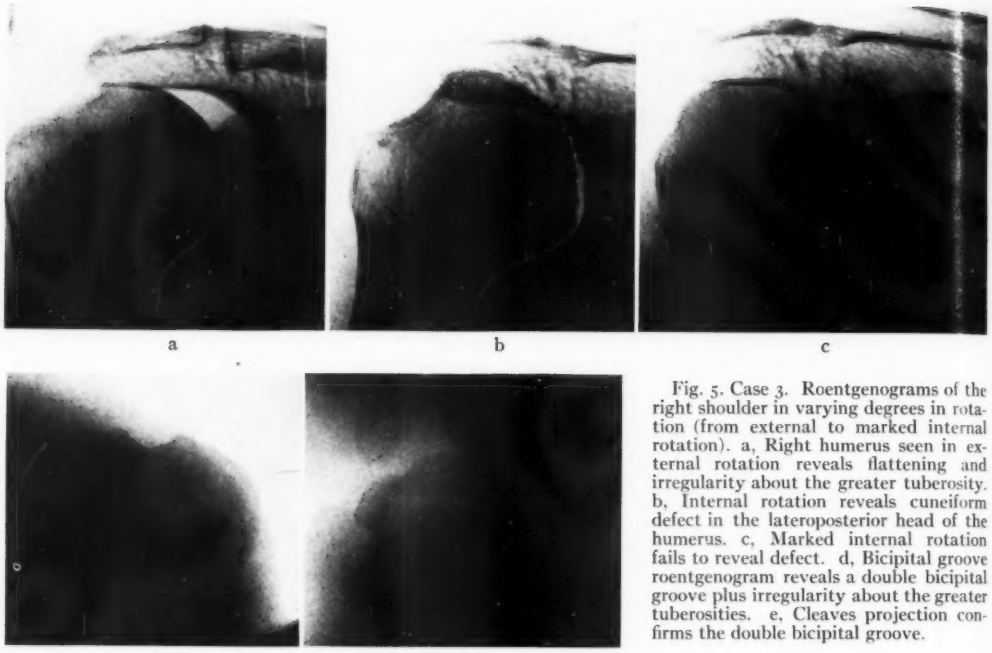


Fig. 5. Case 3. Roentgenograms of the right shoulder in varying degrees in rotation (from external to marked internal rotation). a, Right humerus seen in external rotation reveals flattening and irregularity about the greater tuberosity. b, Internal rotation reveals cuneiform defect in the lateroposterior head of the humerus. c, Marked internal rotation fails to reveal defect. d, Bicipital groove roentgenogram reveals a double bicipital groove plus irregularity about the greater tuberosities. e, Cleaves projection confirms the double bicipital groove.

is our belief that the mechanism of a dislocation can be demonstrated satisfactorily by means of roentgenograms and, at times, fluoroscopy (Figs. 3, 4, 5, 6, 7, 8, 9, 10). The arm is abducted and internally rotated. Trauma is transmitted along its longitudinal axis, forcing the head inferiorly under the inferior lip of the glenoid (subglenoid dislocation). The lateroposterior portion of the humeral head impinges on the glenoid lip as it is forced anteriorly through the capsule into the axilla. The inferoanterior lip of the glenoid is very frequently fractured at this stage. Approximately one-half of the so called subcoracoid dislocations are actually inferoanterior or axillary dislocations. The other half follows the path of least resistance anteriorly and superiorly to become true subcoracoid dislocations. Beside its academic value, the latter statement has very little significance as both the axillary and subcoracoid dislocations are reduced in a similar way.

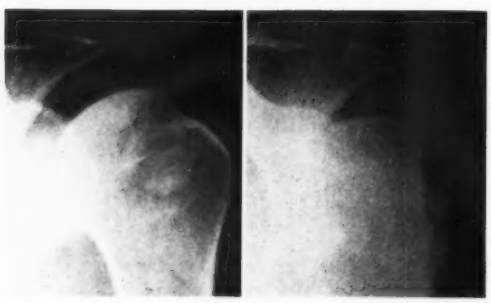
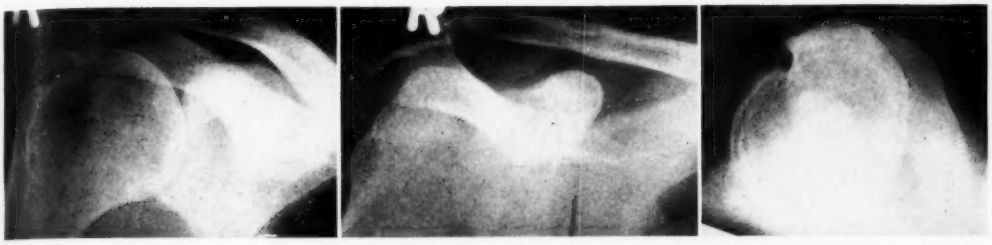


Fig. 6. Case 4. a, left, External rotation. The bone island seen in the region of the greater tuberosity is of doubtful significance. b, Internal rotation reveals a shearing fracture of the lateroposterior head of the humerus with two small bone fragments.

The degree of internal rotation determines whether the glenoid lip will impinge on the lateroposterior aspect of the humerus with a resultant compression fracture (defect) of which the thickened border is actually compressed spongiosa bone (7). If the arm is in partial internal or even external rotation, the glenoid lip will impinge on the greater tuberosity producing the typical avulsion fracture (Figs. 8, a and 9, a). Less often cystic changes may develop in the lateroposterior part of the spongiosa of the head of the humerus. They are small and sharply circumscribed areas of rarefaction. These are due to small compression fractures or to aseptic necrosis as a result of traumatic disturbance of the vascular supply to this region (Fig. 10, a, b, c, d).



a b c

Fig. 7. Case 5. a, Right humerus in external rotation reveals minimal changes in the region of the tendinous inserion of the superspinatus which may be due to tears. b, Internal rotation reveals shearing fracture of the lateroposterior head of the humerus. c, Bicipital groove projection reveals spur formation overlying the bicipital groove.

In view of our studies of the mechanics of inferoanterior dislocations, it becomes impracticable to explain the grooved defect or other bony changes in the humeral head on the basis of such theories as congenital anomalies, osteochondritis dissecans, posttraumatic inflammation or leverage against the acromion. In spite of the many fractures of the greater tuberosity, we have never seen a fracture of the acromion associated with an inferoanterior dislocation.

Anatomical roentgenological appearance of the defect. The defect is located posterior and medial to the greater tuberosity on the lateroposterior aspect of the articulating head (Fig. 2, a, b). It is cuneiform or navicular in shape and its size varies with the diameter of the head and trauma (4, 5). Average dimensions are 2.5 centimeters in length, 1.5 centimeters in width, and 0.75 centimeters in depth. The defect is demarcated from the surrounding normal bone by sharp or vertical projecting walls which are at right angles to each other. The spongiosa bordering the defect is compressed and therefore much thicker than usual.

Routine films of the shoulder with the arm held in adduction and external rotation fail to reveal the defect. In external rotation, rarefaction may be seen in the region medial to the greater tuberosity. In large defects, some flattening of the lateral contour of the articular surface may be existant. The defect is seen best with the arm in internal rotation, bringing that portion of the humerus into profile view. A sharp vertical line of condensation extending downward from the top of the head of the humerus, parallel and slightly lateral to the shaft axis, comprises the medial border of the defect. This line of condensation is the result of spongiosa bone compression. In recurrent dislocations, the defect may increase in size and the borders become more sclerotic in appearance. Probably for this reason, earlier surgeons called these defects congenital anomalies.

Bicipital groove. When the humeral head forces a rent in the anterior portion of the capsule, it is

a b c

Fig. 8. Case 6. a, Subglenoid dislocation, external rotation, with avulsion of the greater tuberosity. b, Fragment in position immediately after correction of dislocation. c, Re-examination 18 months later reveals complete absorption of the greater tuberosity (Courtesy, *Radiology*, December, 1940).

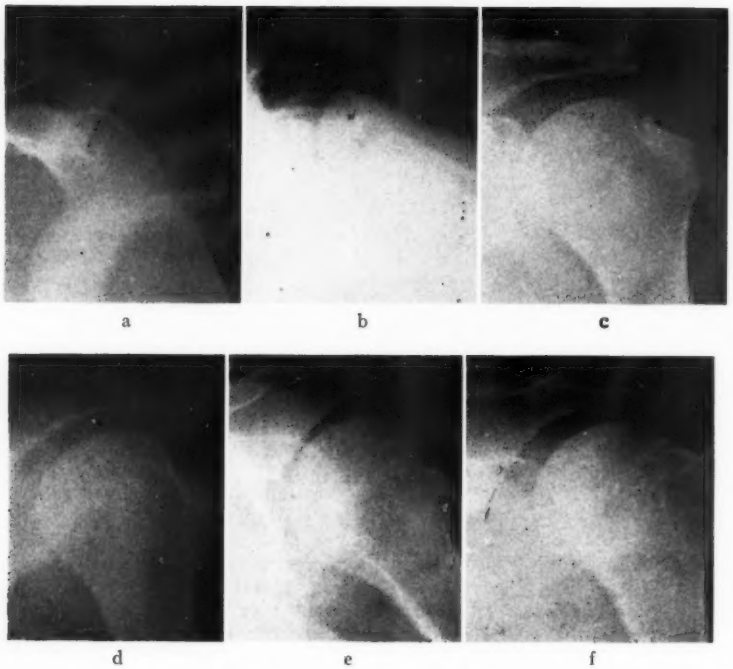


Fig. 9. Case 7. a, Subglenoid dislocation in partial external rotation with avulsion fracture of the posterior portion of the greater tuberosity. b, Dislocation has been corrected with fragment in good position. c, Examination 3 months later reveals some depression in the region of the greater tuberosity. d, Examination 6 months later reveals cuneiform defect in the lateroposterior head of the humerus. e, Re-examination 1 year later reveals no change in the defect in the lateroposterior head of the humerus. f, External rotation fails to reveal the defect.

reasonable to assume that the bicipital tendon is also injured. We have developed the following technique for visualization of the bicipital groove (10): the arm is adducted to the side of the body in supination (external rotation); the tube is placed slightly external to and below the level of the elbow and the central ray is directed at a tangent upward and medial toward the groove in the humeral head. The casette is placed on top of the shoulder and at right angles to the longitudinal axis of the humerus (Fig. 11, a, b).

Tangential projection studies of normal shoulders reveal a smooth notch in the center of the humerus which is the bicipital groove (Fig. 12, a). On each side of the groove are smooth prominent walls of the lesser and greater tuberosities. The lesser tuberosity is medial and its wall is more perpendicular than that of the greater tuberosity.

Changes as follows may be observed in shoulders with histories of previous trauma whose routine roentgenograms are within normal limits.

1. Scalloping and irregularity of the groove which may account for tendon fraying (sometimes a small osteophytic spur, 1 to 4 millimeters in length is noted projecting into the groove).

2. Presence of one or more additional grooves due to tendon dislocations.

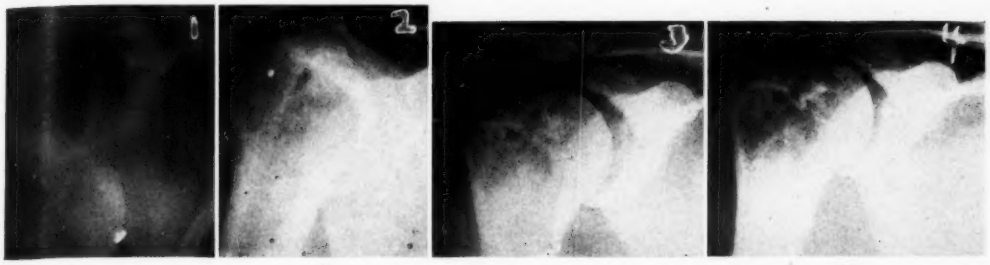
3. Formation of new bone in old groove as result of new groove. Shallow old groove as a result of bicipital tendon disuse when the tendon dislocates and forms a new groove.

CASE REPORTS

CASE 1. J. L. B., male, aged 24 years, injured his left shoulder as a child. Examination revealed a snapping friction noise as the shoulder was pushed forward by a voluntary motion. Clinically there appeared to be an anterior subluxation caused by rubbing of the humeral head on the anterior cartilaginous portion of the glenoid rim. However, roentgenograms in both positions revealed that instead of being an anterior subluxation, it actually was a subglenoid subluxation (Fig. 4, a, b).

CASE 2. H. C., male, aged 22 years, injured his right shoulder many years prior to admission when he struck another person. Since that time he has had repeated dislocations even at the slightest efforts, such as raising a chair from the floor. He reduced the luxation himself by the Kocher maneuver. Roentgenograms in marked extension revealed that not only was there a partial subglenoid subluxation of the right shoulder but also, when left humerus was extended, a corresponding subluxation (Fig. 4, c, d).

This case exemplifies the need of individualizing each case before attempting therapy. In this case a Nicola operation on the right shoulder had been done and as might have been anticipated, the patient immediately redislocated his shoulder when his arm was removed from the sling.



a b c d

Fig. 10. Case 8. a, Axillary dislocation right humerus. b, After correction of the dislocation there is rarefaction in the lateral portion of the humeral head. c, Re-examination 3 months later reveals moderate flattening of the greater tuberosity with multiple circular areas of rarefaction in the lateral humeral head. d, Examination 6 months later reveals increase in multiple areas of rarefaction in the lateral portion of the humeral head.

CASE 3. J. Mac., male, aged 29 years, stated that at the age of 10 years he fell off an acrobatic bar and was temporarily unconscious. He did not know whether he had had a dislocation. In 1935, he was troubled with neck pain and later a similar pain occurred in his left shoulder. His shoulder joints were stiff and fixed in an adducted position. However, he had been getting more motion back into the joint. Roentgenograms taken on August 7, 1941, in external rotation revealed minimal rarefaction in the lateral portion of the humerus and some tendency toward flattening of the greater tuberosity. In moderate internal rotation, a moderate size defect was seen in the lateroposterior head of the humerus. In marked internal rotation, the defect was not seen (Fig. 5). Bicipital groove projection revealed irregularity and a double groove (Fig. 5, d). Cleaves projection confirmed the double bicipital groove as a result of previous trauma and probable dislocation of the tendon (Fig. 5, e).

This case illustrates the fact that the defect or fracture will occur on that portion of the humerus which comes in contact with the glenoid lip at the time of dislocation. The contact points between the humerus and the glenoid lip are determined by the amount of rotation of the humerus. In external rotation the greater tuberosity will be involved. In complete internal rotation the lateroposterior head of the humerus will be involved.

The following case illustrates the shearing force the glenoid lip may have when it is impinged on by the humeral head.

CASE 4. J. L., male, aged 40 years, chronic alcoholic, stated that he had had at least 20 dislocations of both shoulders. Roentgenogram of the left shoulder in external rotation revealed nothing unusual, except possibly a small circular area of increased bone density about 2 centimeters in diameter in the midportion of the humeral head which might have been attributed to bone island (Fig. 6, a). However, the film in internal rotation revealed a shearing fracture of the posterior head of the humerus with two residual bone fragments (Fig. 6, b).

CASE 5. J. M., male, aged 28 years, dislocated his right shoulder approximately 1 year prior to his entrance to the hospital. Dislocation was corrected. Subsequently the patient complained of pain in this shoulder on movement. In external rotation, roentgen examination was negative (Fig. 7, a). Internal rotation revealed a shearing fracture of the lateroposterior head of the humerus (Fig. 7, b). Bicipital groove film revealed osteophytic changes overlapping

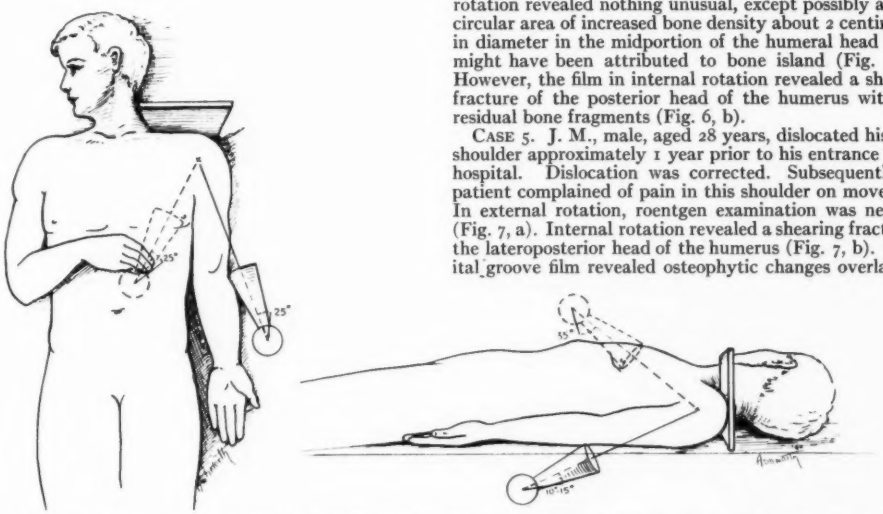
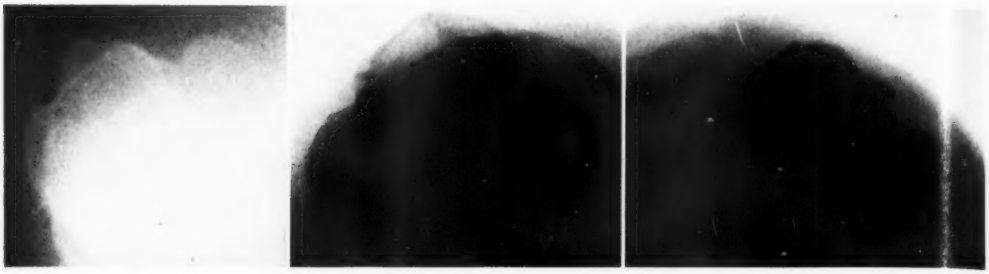


Fig. 11. Diagrams illustrating bicipital groove technique. Dotted line technique has been discarded as impracticable. (Courtesy *Radiology*, June, 1941.)



a b c

Fig. 12. a, Roentgenogram of normal groove. Smooth indentation is the bicipital groove. Lesser tuberosity is more prominent than the greater. b, Case 9. Bicipital groove projection right shoulder reveals tendency toward spur formation on the lesser tuberosity overlying the groove with several circular areas of rarefaction in the tuberosities. c, Case 10. Bicipital groove projection of the left shoulder reveals scallop formation over the lesser tuberosity plus tendency toward spur formation of the lesser tuberosity bridging the bicipital groove.

the groove (Fig. 7, c). These changes probably account for pain on movement.

CASE 6. J. J., male, aged 50 years, had a left subglenoid dislocation on August 14, 1937 (Fig. 8, a, b). The patient was not seen for 1 year and subsequent examination and films on February 14, 1939, revealed that the large avulsion fragment of the greater tuberosity was completely absorbed and there was a marked loss of function of the shoulder primarily limited to abduction and rotation (Fig. 8, c).

CASE 7. E. B., female, aged 50 years, dislocated her left shoulder. Roentgenogram revealed an inferoanterior dislocation of the humerus in partial internal rotation with an avulsion fracture of the posterior portion of the greater tuberosity as well as the lateroposterior portion of the humeral head (Fig. 9, a). Subsequent films taken over a period of 1 year revealed gradual absorption of the fragments with a resultant smooth defect about three-fourths centimeter in diameter in the lateroposterior portion of the humeral head; there was no evidence of fragmentation (Fig. 9, b, c, d, e, f). The shoulder function was within normal limits. Apparently the external rotator muscles were not involved.

CASE 8. W. H., male, aged 30 years, gave a history of dislocating his right shoulder on April 30, 1938, with typical inferoanterior dislocation (Fig. 10, a, b). Subsequent films taken July 12, 1938, and October 10, 1938, revealed marked increase in rarefaction in the lateral position of the head of the humerus with tendency toward compression defect (Fig. 10, c, d).

CASE 9. K. C. M., male, aged 66 years, complained of pain on movement of the right shoulder. There was no history of previous trauma. Routine films in external and internal rotation were negative. Bicipital groove film revealed minute areas of rarefaction bordering the groove with a tendency toward osteophyte formation on the medial border of the groove (Fig. 12, b).

CASE 10. C. B., male, aged 65 years, gave a history of being thrown from a horse 32 years ago. After that time the patient complained of occasional pain in the shoulder joint with minimal limitation of motion. Examination of the left shoulder on February 9, 1942, revealed a moderate atrophy of the left deltoid and pectoral muscles. There was moderate limitation of the humerus to abduction and external internal rotation. Routine films in external and internal rotation were negative. Bicipital groove roentgenograms revealed a tendency toward scallop formation on the lesser tuberosity with an attempt at spur formation of the lesser tuberosity bridging the bicipital groove (Fig. 12, c).

SUMMARY AND CONCLUSIONS

1. Thorough anatomical knowledge is a requisite to better understanding of complications in shoulder dislocations as shown by roentgenograms.

2. Mechanics of dislocations as previously taught is apparently erroneous and more careful study should be given to the mechanical anatomy in order to apply suitable therapeutic measures in correcting and preventing recurrent dislocations.

3. Defect in the lateroposterior head of the humerus, avulsion fracture of the greater tuberosity, fractures of the inferoanterior glenoid lip, and cystic areas in the lateral head of the humerus are some of the complications associated with inferoanterior dislocations of the humerus. (a) In cases of posttraumatic pain, bicipital groove roentgenograms should be taken when routine films fail to reveal any evidence of abnormality. (Routine films include one film in external rotation, a second in extreme internal rotation and a third in lateral view.)

Far too often posttraumatic pain is attributed to a psychotic state or anticipation of compensation on the part of the patient.

REFERENCES

1. CLEAVES, E. N. Am. J. Roentg., 1941, 45: 288–295.
2. CODMAN, E. A. The Shoulder. Boston: Thomas Todd, 1934.
3. HERMODSSON, I. Acta radiol., 1934, Supp. 20, 1–173.
4. HILL, H. A., and SACHS, M. D. Proc. Third Congress Pan-Pacific Surg. Ass., Honolulu, 1939. September, 316–324.
5. Idem. Radiology, 1940, 35: 690–700.
6. HOBART, M. H. Am. J. Surg., 1939, 43: 279–286.
7. MEYER, A. W. Personal communication.
8. NICOLA, T. J. Bone Surg., 1934, 16: 663–670.
9. PILZ, W. Arch. klin. Chir., 1925, 135: 1–22.
10. SACHS, M. D., HILL, H. A., and CHUINARD, E. L. Radiology, 1941, 36: 731–735.
11. SCHULTZE, E. Arch. klin. Chir., 1914, 104: 138–179.
12. SNODGRASS, L. E. Ann. Surg., 1934, 100: 539–543.

BEADED WIRES IN CLOSED REDUCTION OF FRACTURES OF THE LEG

CHARLES N. PEASE, M.D., F.A.C.S., Chicago, Illinois

WITH the introduction of so called nonirritating metals there has been a great increase in the number of open reductions of fractures of the leg. Simultaneously, there has been a condemnation by a few authors of all closed methods of reduction of fractures of the tibia (1, 2, 3). The blanket indictment of the closed method is unfair as it is still most desirable, and it is hoped this paper will prove such claims and offer a simple positive method for handling fractures of the leg.

A true transverse fracture of the tibia usually can be reduced by manipulation and held in a plaster cast, allowing early weight bearing. However, such management is not often possible in spiral, oblique, and comminuted fractures. To overcome the difficulties in maintaining the position of these fragments, beaded Kirschner wires have been employed in conjunction with closed reduction. The beaded wire was designed by Drs. J. E. M. Thomson and C. F. Ferciot and consists of a metal bead five thirty-seconds of an inch in diameter which has been brazed on to a plain Kirschner wire. Thomson and Ferciot used these wires in fixation of fractures after reduction had been accomplished by open operation, but in my experience open reduction of fractures of the shaft of the tibia has not been necessary.

Closed reduction of fracture of the shaft of the tibia with the use of the beaded wires has been used in 19 cases, 7 of which had been compounded.

Presented as part of the Fracture Course of The American Academy of Orthopedic Surgeons, Atlantic City, New Jersey, January 11, 1942.

METHOD

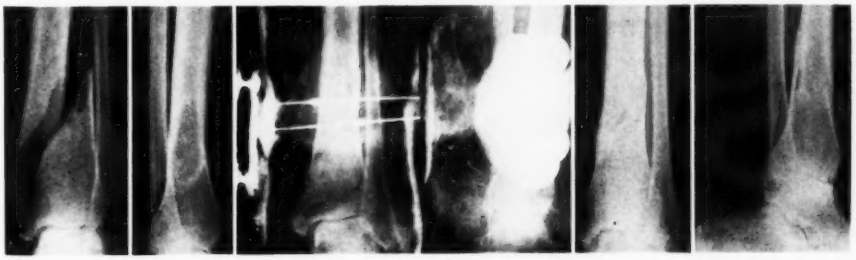
Before the initial steps in reduction are undertaken, a careful study of the roentgenograms is most important. The reason for this is to determine the shape, position, and number of the fragments to ascertain the direction of insertion of the beaded wires.

With the patient on a fracture table and under general anesthesia, a plain Kirschner wire is drilled through the os calcis. A bow is then applied and fastened to the foot piece of the fracture table after which screw traction is applied until the fragments can be gently manipulated into position.

The site of election for drilling one beaded wire can be easily determined by palpating the end of a long fragment aided by the knowledge received from the roentgen examination. However, the area may be selected by use of the fluoroscope.

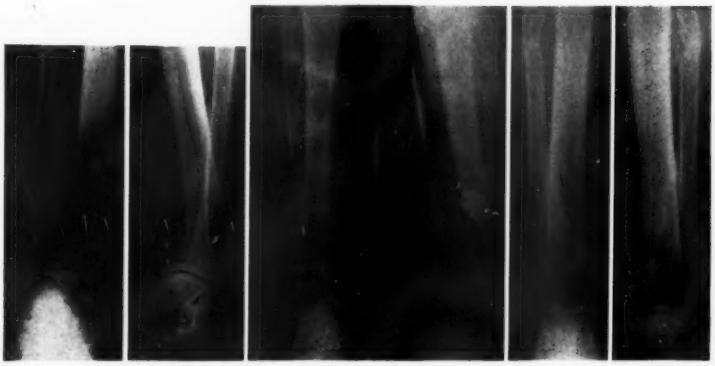
The beaded wire is then drilled into the bone. A puncture wound must be made in the skin to admit the bead, but it is not necessary or recommended that the incision be any deeper than the skin. When the bead strikes the bone, further progress of the wire is naturally stopped thus indicating satisfactory insertion. With this wire acting as a landmark the remaining beaded wire or wires to be drilled are introduced alternately in opposite directions.

A Kirschner bow with a Thomson attachment or the double transverse traction bow is then applied and the fragments are brought together (Fig. 8). A long leg cast is applied and after it has set tautening bolts (5) are fastened to the protruding wires in order to hold the beads firmly against



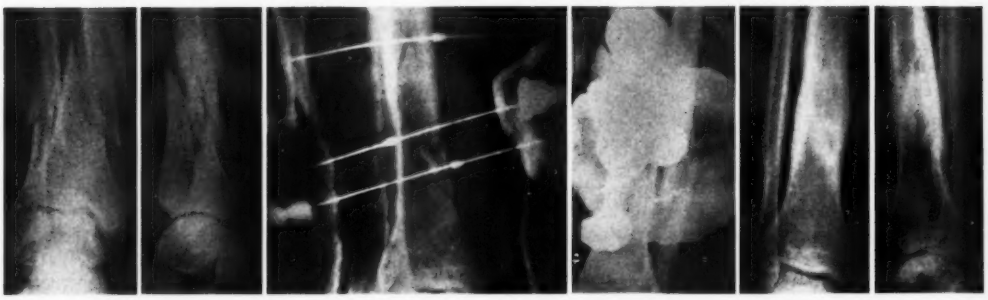
a b c

Fig. 1. Female, aged 49 years. a, Oblique fracture of tibia; b, reduction—2 beaded wires; c, end-result.



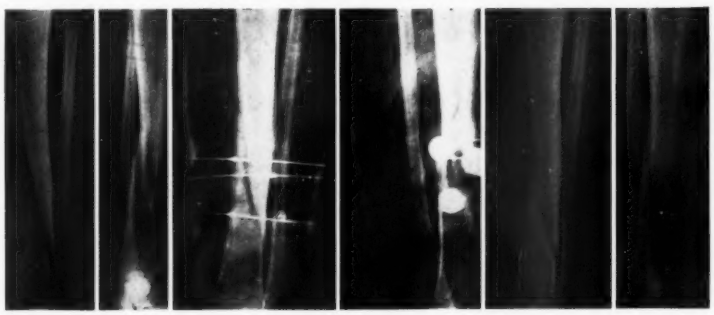
a b c

Fig. 2. Female, aged 58 years. a, Spiral fracture of tibia with fracture of fibula; b, reduction—2 beaded wires; c, end-result.



a b c

Fig. 3. Female, aged 41 years. a, Comminuted spiral fracture of tibia and fibula; b, reduction—3 beaded wires; c, end-result.

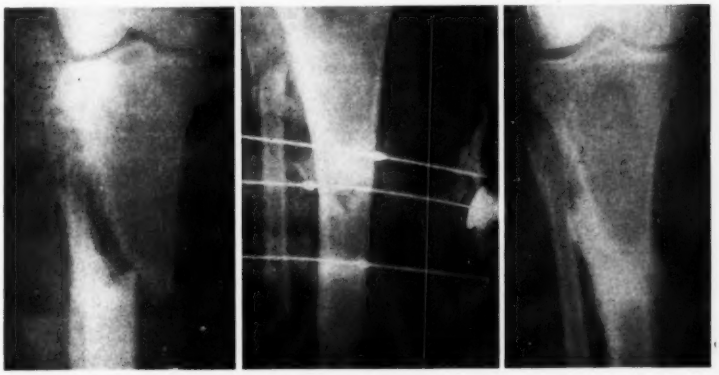


a b c

Fig. 4. Male, aged 43 years. a, Comminuted spiral fracture of tibia; b, reduction—3 beaded wires; c, end-result.

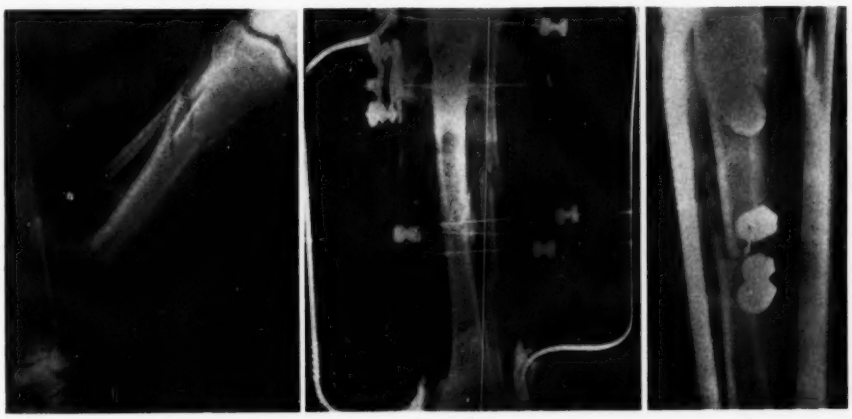
the bone. A washer or slotted metal piece may be placed between the bolt and the cast so that the latter will not become indented.

A long metal "U" shaped bar in which small holes have been drilled at quarter-inch intervals is incorporated in the cast for use in compound frac-



a b c

Fig. 5. Female, aged 40 years. a, Bumper fracture of tibia and fibula unsuccessfully reduced and in a cast; b, reduction—3 beaded wires; c, end-result.



a b c

Fig. 6. Male, aged 58 years. a, Compound fracture of tibia and fibula with massive loss of soft parts; b, reduced—5 beaded wires 3 of which are in turn wired to a plain "U" bar incorporated in the cast.

tures (Fig. 7). The wires are then directed through one of the holes and the tautening bolts are attached to the wires on the outside of the bar. By this means it is not necessary to use counter pressure on the cast by the bolt which allows the removal of the plaster over the denuded area so that the wound may be dressed.

After reduction roentgenograms are made for confirmation of position and, if there has not been adequate transverse traction at the time of reduction, the slack can be taken up by the screw in the tautening bolt.

It is not necessary to apply skin-tight or snug fitting casts, but on the contrary a generous amount of padding is recommended which allows for possible swelling of the extremity. The quantity of sheet wadding used does not interfere with the immobilization of the leg. Although the function of the cast is to support the extremity, this is not of primary importance. The salient point is that it serves as a reinforcement against which the tautening bolts maintain continuous opposing traction of the wires. The fixation, thus being against the bone, the cast is not necessary for maintenance of position of the fragments.

After recovery from the anesthetic the patient may be ambulatory and leave the hospital. Weight bearing with crutches is permitted. After 5 or 6

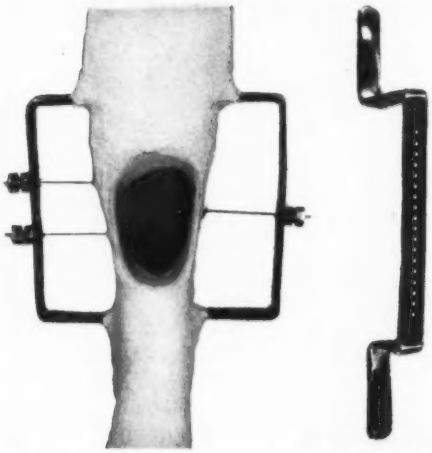


Fig. 7. Cast with perforated "U" bar against which tautening bolts are placed, thus dispensing with secondary wiring to bar as in Figure 6b and the perforated "U" bar.

weeks the cast and wires are removed, and a snug fitting cast is applied allowing full weight bearing as soon as the cast is dry. A block of rubber or a ball of plaster is incorporated in the bottom of the cast to facilitate walking. The plaster ball is recommended because it is harder than the rubber and gives a firmer impact with each step, thus giving more stimulus to new bone formation.

Firm union has been obtained in all cases, the casts having been removed in most instances at the end of 12 weeks, the remainder within 16 weeks.

For better control in drilling, the drill was modified by removal of the trombone guide and the shaft was drilled to accept the wire so that the latter could be fed fractionally into the bone releasing the Jacobs chuck after each 2 inches' insertion (Fig. 9).

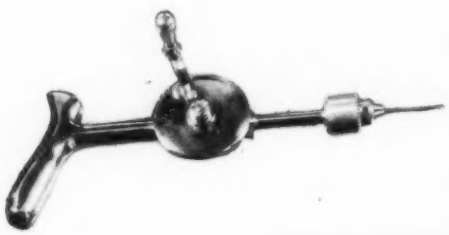


Fig. 9. Kirschner wire drill with trombone guide removed and handle shaft drilled out to accommodate wire.

Fig. 8. Double transverse traction bow.

ADVANTAGES OF METHOD

A very simple procedure is herewith presented which solves a somewhat difficult problem in fractures of the tibia. The advantages of this method are manifold. In the first place, no operation is necessary which at once eliminates elaborate preparation of surgical procedures including necessity for trained assistants. Open operation adds to an already disrupted continuity of soft tissue structures at the site of the fracture by more division of soft tissues which insults the local blood supply so vital for healing and which often interferes materially with vascular drainage of the leg, especially the lymphatic circulation.

It is possible for the patient to leave the hospital as soon as he recovers from the anesthetic. Therefore, he is ambulatory and is allowed guarded weight bearing on the cast. The importance of this cannot be overemphasized. The physiology of a person who is confined to bed is entirely different from one who is up and around. The patient with a fracture who is ambulatory comes nearer to having normal physiological functions which is important to healing of any tissue, especially bone. Early weight bearing is allowed with an attempt to approach as nearly as possible normal local physiology maintaining satisfactory metabolism of bone, keeping demineralization at a minimum and favoring new bone formation. Obviously, this method is ideal for the aged.

In other methods distraction not infrequently occurs; namely, on occasions when screws and plates become loose, under conditions where pins are inserted above and below the fracture site and with the use of longitudinal skeletal traction. Transverse skeletal traction on beaded wires applied in opposite directions with provisions for additional traction any time after the cast is applied holds the fragments firmly against each other insuring bone to bone contact. This factor eradicates unfavorable results from distraction in either the longitudinal or transverse plane, which is a

most important factor in preventing delayed union. In other words positive local apposition is applied continuously. In this series of 19 cases there has been no delayed union. The youngest patient was 17 years, the oldest, 72 years of age.

Closed treatment of fracture of the shaft of the tibia with the use of beaded wires is notable for the little time required to accomplish reduction, for its simplicity and efficiency. Important, also, is that no metal is left in the bone permanently.

REFERENCES

1. HUDACK, STEPHEN. J. Bone Surg., 1941, 23: 896.
2. MANSFIELD, ROBERT D. J. Bone Surg., 1941, 23: 910.
3. MURRAY, C. R. Am. J. Surg., 1941, 51: 739.
4. THOMSON, J. E. M., and FERCIOT, C. F. Surg. Gyn. Obst., 1937, 64: 831.
5. THOMSON, J. E. M., and FERCIOT, C. F. J. Bone Surg., 1936, 18: 245.

COMBINED SPINAL AND INTRAVENOUS ANESTHESIA FOR OPERATIONS ON THE COLON

BRUCE M. ANDERSON, M.D., LLOYD H. MOUSEL, M.D., M.S. (Anes.), and CHARLES W. MAYO, M.D., M.S. (Surg.), F.A.C.S., Rochester, Minnesota

THE term "combined anesthesia" refers to the use of two or more anesthetic agents in conjunction, either simultaneously or in succession but almost always in such a manner that the effect of one overlaps that of the other. This method should employ the beneficial factors of each agent and minimize its inimical ones. This paper, however, concerns certain specific combinations of anesthetics. The development of this method (5, 6) has, of course, paralleled the development of new and better anesthetic agents and methods.

Of the various forms of anesthesia, the one that most often needs the balancing effect of supplementary amnesia, analgesia, or anesthesia (excluding premedication) is spinal anesthesia. This is the form of regional anesthesia whose value is most widely recognized, but Maxson and Weinstein and other authors have ably described the pathological physiology of spinal anesthesia and pointed out its dangers. Cognizant of these dangers, many anesthetists are loathe to employ adequate doses of the anesthetic and a sufficiently high level of anesthesia. As a consequence, they are forced to supplement inadequate spinal anesthesia at a time when, as Wiggin pointed out, "the anesthesia is wearing off and the patient's reflexes are being stimulated from the site of operation," resulting in a stormy induction and poor anesthesia, "with the necessity of a supersaturation of the inhalant anesthetics at the expense of the metabolic requirement of oxygen."

From the Section on Anesthesia, Mayo Foundation and Mayo Clinic, and the Division of Surgery, Mayo Clinic.

Wiggin further said that "because of this difficulty the fear of not obtaining complete height and duration of spinal anesthesia with minimum doses of the agent" has led many anesthetists to the other extreme "of using the maximum dosage of the spinal agent for prolonged spinal anesthesia." Thus vital centers have been encroached upon and the patient's safety and comfort jeopardized.

Realizing this shortcoming of spinal anesthesia, Wiggin, Sankey and Potter, Lorhan and Orr, Woodbridge, Maxson, Weinstein, and Dodd and Hunter advocated combining spinal anesthesia with inhalation anesthesia. Cyclopropane was the unanimous choice for administration by inhalation for the following reasons: (1) it is a potent agent and permits easy induction of anesthesia, and (2) a high concentration of oxygen can be used with it. Some of these authors (3, 4, 14) started the administration of the inhalation anesthetic immediately after the injection of the spinal anesthetic but others (13, 15) used inhalation anesthesia only when necessary. Most of the authors advocated the administration of high concentrations of oxygen.

The introduction of ultrashort-acting barbiturates has made it possible to use intravenous anesthesia in conjunction with spinal anesthesia. Kees apparently was the first to report the use of intravenous anesthesia in conjunction with spinal anesthesia. In the 50 cases which he reported, evipal soluble or pentothal sodium was the anesthetic administered intravenously. This author employed the intravenous anesthesia for the fol-

lowing purposes: (1) to quiet apprehensive patients so that spinal anesthesia might be induced, (2) to supplement inadequate spinal anesthesia, (3) to quiet patients who became apprehensive or nauseated during operation, and (4) to prolong anesthesia.

Sibley and Tuohy also recently have advocated the use of intravenous anesthesia in conjunction with spinal anesthesia. Sibley employed pentothal sodium as the intravenous anesthetic. He used a Y-shaped tube so that physiological saline solution might be administered continuously. The solution of pentothal sodium was administered by means of a 30 cubic centimeter syringe, which was attached to the other arm of the Y tube. This permitted the author to administer the solution of pentothal sodium intermittently in order to keep the patient anesthetized or in a semistuporous state throughout the operation.

In the hands of an experienced anesthetist, intravenous anesthesia and cyclopropane inhalation anesthesia probably are equally satisfactory for use in conjunction with spinal anesthesia. Both of these methods permit the administration of an abundant amount of oxygen and both supplant the deficiencies of spinal anesthesia. In fact, 100 per cent oxygen can be administered in cases in which pentothal sodium is administered intravenously. According to Wiggin, balanced spinal anesthesia possesses the following advantages: (1) the patient is more completely under the control of the anesthetist, (2) the psychic factors of apprehension, restlessness and undue stimulation are completely allayed, (3) respiration can be controlled by the administration of oxygen, (4) the blood pressure and pulse remain more stable than they do with any other method of anesthesia, and (5) complete relaxation can be obtained without the administering of large doses of the spinal anesthetic.

When pentothal sodium and cyclopropane are administered by the intravenous and inhalational methods respectively in conjunction with spinal anesthesia, pentothal sodium has the following advantages: (1) it is nonexplosive, (2) it appeals to the patient as the use of the anesthetic mask can be eliminated, (3) its action is more rapid than that of cyclopropane, (4) it facilitates maintaining a very light plane of amnesia in cases in which this is desirable, and (5) it appears to contribute definitely to the alleviation of postoperative shock.

According to Kees, the barbiturates have certain disadvantages when used in conjunction with spinal anesthesia. Of the disadvantages listed by this author, we may mention the following: (1) singultus occasionally may occur in cases in which evipal soluble is used, (2) the danger of pulmonary complications may be increased, and (3) as barbiturates are detoxicated chiefly in the liver they should not be administered in cases of extensive damage of the liver. Recent investigation (1, 10), however, has demonstrated that pentothal sodium can be administered safely in cases in which severe hepatic deficiency is present. Our experience indicates that the incidence of pulmonary complications is no greater in cases in which pentothal sodium is administered intravenously to supplement spinal anesthesia than it is in cases in which inhalation anesthesia is used for this purpose.

The advantages and disadvantages of spinal anesthesia are well known. If the anesthetist is skilled and if safe doses of the anesthetic agent are employed, there should be little danger of respiratory paralysis or of a decrease in the blood pressure, but nausea, vomiting, and retching frequently cannot be prevented or controlled. In certain cases, difficulties arise when it is necessary to prolong the operation or when the operative procedure involves tissues or organs situated above the planned level of anesthesia. These difficulties frequently necessitate the use of supplemental anesthesia but continuous spinal anesthesia has tended to abate the need for concern over duration of anesthesia in long or potentially long operations. However, intravenous anesthesia also may be used advantageously with continuous spinal anesthesia.

In cases in which intravenous anesthesia is used in conjunction with spinal anesthesia, the intravenous anesthetic should be administered cautiously as the spinal anesthesia will have caused some depression and will have increased the susceptibility of the patient to other anesthetic agents. In order to prevent emotional disturbances and nausea, the intravenous anesthetic should be administered before the incision is made.

It has been said (7) that in order to facilitate operations on the colon it is the anesthetist's responsibility to prevent soiling of the peritoneum in so far as it is possible to do so by keeping the patient from straining. Spinal anesthesia is one of his most valuable aids in accomplishing this and if it cannot be accomplished with spinal anesthesia alone other methods must also be used.

During the past 5 years we have been using a combination of spinal anesthesia and intravenous anesthesia for operations on the colon. Pentothal sodium has been administered intravenously and procaine hydrochloride or metycaine has been administered intraspinally.

TABLE I.—CLINICAL DATA IN CASES IN GROUP 1 AND GROUP 2

		In cases in group 1*	In cases in group 2†
Age of patients, years	20 to 29	4	4
	30 to 39	10	11
	40 to 49	20	29
	50 to 59	29	24
	60 to 69	31	30
	70 to 79	6	2
Anesthetic agent administered intraspinally	Metycaine	56	40
	Procaine hydrochloride	44	57
	Nupercaine		3
Anesthetic agent administered intravenously	Pentothal sodium	100	
Anesthetic agents administered by inhalation	Cyclopropane and oxygen, with or without nitrous oxide and carbon dioxide		16
	Cyclopropane, ether and oxygen, with or without nitrous oxide and carbon dioxide		25
	Nitrous oxide and oxygen, with or without carbon dioxide		8
	Nitrous oxide, ether and oxygen, with or without carbon dioxide		45
	Ethylene, nitrous oxide and oxygen		1
	Ethylene, ether, oxygen and carbon dioxide		2
Operative procedures	Exploratory laparotomy	4	
	Loop colostomy	5	3
	Multiple colostomy	2	1
	Transverse colostomy	2	
	Permanent colostomy		4
	Miscellaneous types of colostomy	2	
	Single-barrel colostomy		8
	McBurney colostomy		3
	Colostomy and biopsy		1
	Colostomy and repair of hernia		1
	Ileocolostomy	5	
Operative procedures	Transcolonic polypectomy	4	4
	Combined abdominoperineal resection: First stage	2	1
	Second stage	3	14
	Single stage	27	29
	Plus other procedures	3	4
	Subsacral	1	
	Anterior resection	3	3
	Extraperitoneal resection	13	11
	Resection of ileum, cecum, etc.	7	10
	Resection of right portion of colon	5	
	Resection of transverse colon	5	
	Resection of portion of sigmoid	2	
	Colocolostomy and excision of carcinomatous segment	1	
	Exteriorization	3	3
	Repair of rectal prolapse	1	
Stimulants administered	Ephedrine sulfate: 25 mg. administered subcutaneously before operation	96	74
	50 mg. administered subcutaneously before operation	4	26
	Two doses of 25 mg. each administered during operation	3	1
	Neosynephrin: 0.2 to 0.3 c.c. of a 1 per cent solution administered intramuscularly	1	
Complications	Pulmonary complications: All pulmonary complications	14	14
	Pneumonia	5	6
	Excluding pneumonia	10	9
	Nausea	10	38
	Vomiting	8	36
Blood and fluids administered intravenously after operation	Blood transfusion	52	34
	Physiological solution of sodium chloride or solution of dextrose or acacia	48	27

*One hundred cases in which intravenous anesthesia was used in conjunction with spinal anesthesia.
†One hundred cases in which inhalation anesthesia was used in conjunction with spinal anesthesia.

The following technique has been employed. The spinal anesthetic is injected before the patient is placed on the operating table. The dose of the spinal anesthetic is the same as that employed in a similar case in which the resulting anesthesia is not to be supplemented with intravenous anesthesia; that is, the dose should be large enough to produce anesthesia of sufficient depth and duration to permit the performance of the contemplated operation. A dose of 25 milligrams of ephedrine sulfate is administered subcutaneously when the spinal anesthetic is administered; in some cases we have administered initial doses of 50 milligrams of ephedrine sulfate.

The patient then is placed on the operating table. After the patient has been draped, towels are clipped to the skin. If this does not cause any pain, one may assume that the spinal anesthesia is effective. A 2.5 per cent solution of pentothal sodium then is injected intravenously. When the

TABLE —II. AVERAGE BLOOD PRESSURE IN CASES IN GROUP 1 AND GROUP 2

	Group 1*			Group 2†		
	Cases	Blood pressure, mm. of mercury		Cases	Blood pressure, mm. of mercury	
		Systolic	Diastolic		Systolic	Diastolic
Before operation	97	129.5	76.6	100	130.6	74.6
Immediately after operation	97	116.1	68.7	99	116.5	63.1
One-half hour after operation	91	114.0	72.5	96	107.5	62.0
One hour after operation	97	125.4	77.9	95	111.7	64.7
First day after operation	87	123.0	72.8	90	123.2	70.5

*One hundred cases in which intravenous anesthesia was used in conjunction with spinal anesthesia.
†One hundred cases in which inhalation anesthesia was used in conjunction with spinal anesthesia.

TABLE III.—PULSE RATE IN CASES IN GROUP 1 AND GROUP 2

	Group 1*		Group 2†	
	Cases	Average pulse rate per minute	Cases	Average pulse rate per minute
Day of operation	100	88.5	100	85.1
One-half hour after operation	87	90.8	93	97.1
One hour after operation	93	89.9	92	93.3
First day after operation	100	92.8	100	98.9
Second day after operation	100	91.9	99	93.8
Third day after operation	100	88.7	99	91.1
Fourth day after operation	99	86.3	99	88.8

*One hundred cases in which intravenous anesthesia was used in conjunction with spinal anesthesia.
†One hundred cases in which inhalation anesthesia was used in conjunction with spinal anesthesia.

injection is begun the patient is told to start counting aloud. The injection is continued until the patient stops counting. A total dose of 5 to 10 cubic centimeters of the solution usually is required to cause the patient to stop counting. The needle is kept in the vein and additional amounts of the solution are injected if necessary. The plane of anesthesia should be just deep enough to prevent the patient from vomiting. A high concentration of oxygen (100 per cent) is administered throughout the operation. If it becomes evident that the desired plane of anesthesia cannot be maintained without the injection of excessive doses of pentothal sodium, a mixture of equal parts of nitrous oxide and oxygen is administered by inhalation in addition to the intravenous pentothal.

If the decrease in the patient's blood pressure becomes severe, 25 milligrams of ephedrine sulfate is injected intravenously or 0.2 to 0.3 cubic centimeter of a 1 per cent solution of neosynephrin is administered intramuscularly. The dose of ephedrine sulfate is repeated once if necessary.

It has been the opinion of one of us (Mayo) that the results obtained in cases in which this method was used have been better than those obtained formerly; that is, when a combination of spinal anesthesia and inhalation anesthesia was being used. In order to determine the relative value of the two methods, we decided to review two comparable groups of cases. Group 1 includes 100 cases in which operations on the colon were performed with spinal anesthesia combined with intravenous anesthesia, and group 2 includes 100 cases in which a combination of spinal anesthesia and inhalation anesthesia was used for similar operations.

The cases in group 2 were observed at an earlier date than were those in group 1. During the period in which the cases in group 1 were observed, it has been our practice to give a transfusion of at least 500 cubic centimeters of blood in the course of, or after the completion of, the operation in all cases in which combined abdominoperineal resection or partial colectomy was performed and in certain other cases in which major operations were performed on the colon. A transfusion of blood was given during or very soon after operation in 51 per cent of all the cases in group 1 (Table I) but in only 34 per cent of the cases in group 2. A blood transfusion was given, or fluids were administered intravenously during or very soon after operation, or both of these procedures were employed in 69 per cent of the cases in group 1 but in only 47 per cent of the cases in group 2. Another difference is that oxygen was administered after operation, by means of a mask or a tent, in a higher percentage of the cases in group 1 than it was in the cases in group 2. The relative influence of these differences in treatment in the two groups of cases will be considered in our analysis of the results obtained.

With the exception of the method of anesthesia employed and the differences in treatment which we have just mentioned, the cases comprising the two groups are essentially similar. The ages of the patients, the anesthetic agents employed, the various operations performed, the stimulants administered and the postoperative treatment em-

ployed are shown in Table I. It will be noted that the ages of the patients were practically the same in each group. The technique of intraspinal injection was the same in all cases. The average amounts of procaine hydrochloride or metycaine injected intraspinally were as follows: 127.9 milligrams in the cases in group 1 and 132.2 milligrams in the cases in group 2.[1] The average total dose of pentothal sodium administered intravenously in the cases in group 1 was 0.919 gram. All of the operations were performed by one of us (Mayo).

The average duration of anesthesia was 69.3 minutes in the cases in group 1 and 66.9 minutes in those in group 2.

The incidence of postoperative pulmonary complications was practically the same in the two groups of cases (Table I). It will be noted that two pulmonary complications, namely, atelectasis and pneumonia, occurred in one case. The corresponding incidence of postoperative pulmonary complications tends to confirm the frequent observation that the incidence of these complications depends upon the type of operation and age of the patient rather than upon the choice of anesthetic agent or method.

The average values for the systolic and diastolic blood pressures, the average pulse rate and the average respiratory rate, as determined before operation and at various intervals after operation, are shown in Tables II, III, and IV, respectively. It will be noted that there was very little difference in the respective findings in the two groups of cases before operation. During the first hour after operation the average decrease in the blood pressure was definitely greater in group 2 than it was in group 1 (Table II). Although this difference in blood pressure may be partially attributable to increased use of blood transfusion and the intravenous administration of fluids in the cases in group 1, it is our opinion that much credit must be given to the intravenous administration of pentothal sodium and to the continuous administration of 100 per cent oxygen.

In Table II it will be noted that immediately after operation the average decrease[2] in the systolic blood pressure was greater in the cases in group 2 than it was in the cases in group 1. Half an hour after operation, the average decrease in the systolic pressure was 15.5 millimeters in group 1 and 23.1 in group 2. One hour after operation the average decrease was 4.1 millimeters in group 1 and 18.9 millimeters in group 2. However, the average decrease during the first day after operation was approximately the same in the two groups of cases, that is, 6.5 millimeters in group 1 and 7.4 millimeters in group 2.

[1]As there is a difference of only 10 per cent in the doses of these drugs, the doses have been averaged together.

[2]The average decrease in the blood pressure is based on the reading obtained before operation.

TABLE IV.—RESPIRATORY RATE IN CASES IN GROUP 1 AND GROUP 2

Time after operation	Group 1*		Group 2†	
	Cases	Average respiratory rate per minute	Cases	Average respiratory rate per minute
One-half hour	86	20.9	90	21.2
One hour	94	20.8	91	20.5
First day	100	19.5	95	20.5
Second day	99	20.1	94	20.6

*One hundred cases in which intravenous anesthesia was used in conjunction with spinal anesthesia.
†One hundred cases in which inhalation anesthesia was used in conjunction with spinal anesthesia.

As was to be expected, there was a concomitant rise in the average pulse rate after operation (Table III). Half an hour after operation, the average increase in the pulse rate per minute was 2.3 in the cases in group 1 and 12 in the cases in group 2. One hour after operation, the respective increases were 1.4 and 8.2. When blood pressure falls, the expected physiological response is an increase in the pulse rate. This response was observed in both groups of cases but on the first day after operation, when the average decrease in the systolic blood pressure was approximately the same in the two groups of cases, that is, 6.5 millimeters in group 1 and 7.4 millimeters in group 2, the average increase in the pulse rate per minute was 4.3 in group 1 and 13.8 in group 2. This variation of the difference in the average pulse rate in the two groups of cases persisted for the first 4 days after operation. This indicates that the general condition of the patients in group 2 was less satisfactory than was the condition of the patients in group 1. We believe that the more favorable response of the pulse rate in group 1 is attributable to the use of pentothal sodium and to the improvement in the treatment employed immediately after operation.

The important differences in the two series of cases were in the incidence of nausea and vomiting (Table I), in the duration of the period of recovery, and in the mortality rate. In group 1, 10 per cent of the patients had nausea during or after operation and 8 per cent vomited, while in group 2, 38 per cent of the patients were nauseated and 36 per cent vomited. Such a reduction in the incidence of nausea and vomiting is of great importance not only from the standpoint of the

patient's comfort but also in facilitating the surgeon's work, prevention of soiling of peritoneum, and lessening the strain upon the wound.

The shorter duration of the period of recovery from anesthesia in group 1 is another difference, which is due almost solely to the use of pentothal sodium with spinal anesthesia. In group 1, the patients were awake on an average of 27 minutes after operation while in group 2 it was 45 minutes before they responded. A shorter period of recovery is of great advantage, of course, in rendering the patient more co-operative and also in allowing a more rapid return to normal of muscle tone and other physiological processes.

However, one of the most striking differences in the two series is in the mortality rate. In group 2, it was 12 per cent, while in group 1, it was only 5 per cent. Whereas we realize that the other differences in the treatment of these two groups of patients are significant, we feel that the combination of intravenous anesthesia with spinal anesthesia is of paramount importance in producing these favorable results.

REFERENCES

1. Anderson, B. M. Unpublished data.
2. Dodd, Harold, and Hunter, J. T. Lancet, 1939, 1: 685–688.
3. Kees, P. A. Anesth. & Analg., 1941, 20: 24–26.
4. Lorhan, P. H., and Orr, T. G. J. Kansas M. Soc., 1940, 41: 13–15.
5. Lundy, J. S. Minnesota M., 1926, 9: 399–404.
6. Idem. J. Lancet, 1931, 51: 743–745.
7. Idem. Personal communication to the authors.
8. Maxson, L. H. Anesth. & Analg., 1938, 17: 61–70.
9. Sankey, B. B., and Potter, J. K. Ohio M. J., 1940, 36: 29–30.
10. Scheifley, C. H., and Higgins, G. M. Am. J. M. Sc., 1940, 200: 264–268.
11. Sibley, W. L. South. M. J., 1941, 34: 910–913.
12. Tuohy, E. B. South. M. J., 1941, 34: 42–45.
13. Weinstein, Mandel. Anesth. & Analg., 1938, 17: 352–356.
14. Wiggin, S. C. Anesth. & Analg., 1939, 18: 193–195.
15. Woodbridge, P. D. Surg. Clin. N. America, 1935, 15: 1513–1525.

INTRAVENOUS AMINO ACID ADMINISTRATION IN SURGICAL PATIENTS USING AN ENZYMATIC CASEIN DIGEST

CLARENCE E. GARDNER, Jr., M.D., F.A.C.S., and JOSIAH C. TRENT, M.D.,
Durham, North Carolina

MAINTENANCE of an adequate protein intake in surgical patients is necessary for proper wound healing, for replacement of tissues lost in metabolism, and for the protection of body tissues, particularly the liver. Because of the relatively small reserve of protein available for this purpose, serious protein deficiencies may develop fairly quickly in those patients in whom feeding by mouth is either impossible or unwise. The frequency and seriousness of these deficiencies, resulting particularly in hypoproteinemia, have been repeatedly emphasized.

Efforts to preserve nitrogen equilibrium by parenteral protein administration have occupied the attention of numerous investigators in recent years, and it has now become possible to maintain nitrogen metabolism either by plasma infusions or by the intravenous administration of amino acids. Plasma transfusion is a life-saving procedure when acute reduction of plasma volume occurs such as in traumatic shock or burns. The use of plasma as a nutritive agent, however, is impracticable because of the large quantity necessary for such a purpose. (In our cases, the quantity of plasma recovered from approximately 2 liters of blood would have been necessary to supply an amount of protein equal to that given each day as amino acid.) Furthermore, plasma proteins are less satisfactory than amino acids for nutrition since they are not available for utilization by the tissues until they have been hydrolized.

Elman and Weiner (7) were the first to give amino acids intravenously to human beings. They demonstrated satisfactorily that a mixture of amino acids derived from the acid hydrolysis of casein to which tryptophan and cystine had been added was utilized by the body.

The mixture of amino acids—amigen—used in this study was prepared by the enzymatic hydrolysis of casein. Tryptophan and cystine are not destroyed by the enzymes and need not be added, thus considerably lowering the cost of manufacture. The mixture is a complete one which is nonantigenic and which, as the sole source of nitrogen in experimental animals, will support a normal rate of growth (17).

From the Department of Surgery, Duke University School of Medicine.

We have administered the amino acid mixture intravenously to a series of surgical patients (chiefly postoperative), receiving no other protein. Included in this series were patients with the following conditions: appendiceal peritonitis, carcinoma of the stomach, gastric resection for duodenal ulcer, perforated peptic ulcer, ruptured ileum, intestinal obstruction, carcinoma of cecum, and polyposis of the colon. In this study it has been our purpose to observe the effect of such injections of amino acid on the clinical course and on the plasma proteins and to determine the safety of the procedure as judged by reactions and complications.

The amino acid mixture has been supplied as a powder or as a sterile 10 per cent solution in liter flasks. When using the powder our practice has been to prepare a 10 per cent stock solution in freshly distilled pyrogen-free water which is then sterilized by passing through a Seitz filter. The solution for intravenous use was prepared by adding 300 cubic centimeters of the 10 per cent stock solution to 700 cubic centimeters of 10 per cent dextrose in saline. When using the 10 per cent solution as supplied by the manufacturer a similar dilution was made. One liter of this 3 per cent amino acid solution was then given intravenously as rapidly as it would pass by gravity through a 20 gauge needle, the average time of injection being about 60 minutes. Two such liter infusions were given daily to the adults in this study while children received 1 liter daily.

REACTIONS

Three hundred and sixty-two intravenous infusions of this mixture have been given to 30 patients as the only source of nutrition over an average period of 7 days. The longest period of administration was 14 days, the shortest 3 days.

We have observed 3 chills which were followed by a temperature elevation and 1 elevation of tem-

TABLE I.—NITROGEN BALANCE STUDIES IN AN AFEBRILE POSTOPERATIVE PATIENT WITH MALFUNCTIONING GASTROENTEROSTOMY STOMA

Postoperative day			4	5	6	7	8	9	10	11
Blood findings	Total protein (gms.)			4.8			5.3			5.4
	Albumin			2.1			2.5			2.6
	Globulin			2.7			2.8			2.8
	Red blood cells			4.24			4.00			3.89
	Hematocrit			34.8			39.2			36.1
Total intake (intravenous)	Glucose (gms.)		140	140	140	140	140	140	140	140
	Vitamin	B_1 (mgm.)	10	10	10	10	10	10	10	10
		C (mgm.)	100	100	100	100	100	100	100	100
		Nicotinic acid (mgm.)	25	25	25	25	25	25	25	25
	Amigen (gms.)		60	60	60	60	55	60	60	30
	Total nitrogen (gms.)		7.2	7.2	7.2	7.2	6.6	7.2	7.2	3.6
Output urine	Total c.c.			2580	3410	2250	2000	2740	2000	
	Total nitrogen			5.0	5.8	4.1	4.0	4.6	4.7	
Nitrogen balance (gms.)				+2.2	+1.4	+3.1	+2.6	+2.6	+2.7	

(A66308)

perature without chill or subjective discomfort. In spite of the fairly rapid rate of injection, we have encountered no nausea, vomiting, abdominal pain, or flushing. Occasionally patients complained of a feeling of warmth during the injection, but this sensation was not unpleasant. After repeated injections of the amino acid solutions into the same vein, thrombosis of the vein usually developed, but by careful selection of the site of injection thrombosis was no more frequent than when plain 10 per cent dextrose solutions were used. Clinically patients who received the amino acids appeared less ill and subjectively felt better than those who had not received them. No reactions have occurred which would indicate that amigen is antigenic.

EVIDENCE OF UTILIZATION

Nitrogen balance studies were carried out in all of the patients studied. In those with low nitrogen outputs nitrogen balance was easily achieved (Table I). In all of the patients with fever and in

TABLE II.—NITROGEN BALANCE STUDIES IN A POSTOPERATIVE PATIENT WITH APPENDICEAL PERITONITIS

Postoperative day			1	2	3	4	5	6	7	8
Blood findings	Total protein (gms.)		5.8		5.2		5.1		5.7	
	Albumin				2.6		2.7		2.9	
	Globulin				2.6		2.4		2.8	
	Red blood cells		4.6		4.55	3.99	4.44		4.2	
	Hematocrit		35.9		33.7	33.2	31.8		33.2	
Total intake (intravenous)	Glucose (gms.)		140	140	140	140	140	140	140	140
	Vitamin	B_1 (mgm.)	10	10	10	10	10	10	10	10
		C (mgm.)	100	100	100	100	100	100	100	100
		Nicotinic acid (mgm.)	25	25	25	25	25	25	25	25
	Amigen (gms.)		60	60	60	60	60	60	60	60
	Total nitrogen (gms.)		7.2	7.2	7.2	7.2	7.2	7.2	7.2	7.2
Output urine	Total c.c.			780	1585	1645	1690	1305	1350	1340
	Total nitrogen (gms.)			9.3	11.0	12.0	20.1	16.3	14.8	17.4
	Amino nitrogen (mgm.)			85.8	110.0		57.0		121.0	

(A76307)

most of the patients immediately following operation large nitrogen excretions (in one case 25 gms. in 24 hours) made it impossible to attain nitrogen balance on the doses of amino acid given (Table II).

Although all injections were given as rapidly as they would pass by gravity through a 20 gauge needle, the amino acids were not spilled into the urine in significant quantities. Estimation of amino nitrogen in 24 hour specimens from most of the patients studied in no case gave readings higher than 400 milligrams of amino acid nitrogen in the urine per day. The average excretion per day was 163 milligrams.

Evidences of serum protein regeneration following intravenous amino acid administration has been proved in dogs by Elman (5) and also by Whipple and his co-workers. In our patients who were not running fever, we were able to demonstrate a rise in serum protein in the blood when amino acids were the sole source of nitrogen intake as shown in Figure 1. On the amount of amino acid used, we were unable to demonstrate a rise in serum protein in those whose courses were febrile.

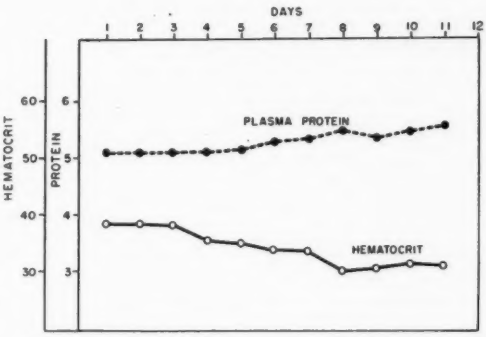


Fig. 1. Chart showing the average daily plasma protein and the hematocrit in 7 afebrile postoperative patients each of whom received 60 grams of amino acid daily as their only source of nitrogen intake.

OBSERVATIONS

Our experience indicates that this amino acid solution prepared by the enzymatic hydrolysis of casein may be given intravenously without significant reaction to surgical patients and that it is retained and apparently utilized by the body. In afebrile patients when it represents the sole source of nitrogen replacement, nitrogen balance has been attained and definite regeneration of plasma protein has been demonstrated.

In all cases adequate amounts of glucose must be given along with the amino acids for their "sparing" action of the protein. Without carbohydrates the protein administered may be used solely for energy and in addition tissue proteins also may be broken down and used for this purpose. With glucose, nitrogen which would otherwise be wasted is retained. In regard to the quantity of amino acid administered, we have arbitrarily given 60 grams of amino acid daily to adults and 30 grams to children. This is more than the minimal basal requirement of protein estimated at 0.5 gram per kilogram but is probably less than the usual needs for postoperative, febrile, or acutely ill patients. Our practice has been to give 70 grams of glucose with each 30 grams of amino acid, this is a minimal amount of glucose to act properly as a sparer of protein. If the glucose is to serve as the only source of energy for the patient larger quantities become necessary

In patients with fever, huge amounts of protein and glucose are necessary if the patient is to be kept in positive nitrogen balance. To prevent wasting of the body tissues in such patients, it may be necessary to give 2 or even more times the quantity of amino acids we have been giving.

SUMMARY

In 30 postoperative patients in whom feeding by mouth was impossible or unwise a solution containing glucose, vitamins, sodium chloride, and amino acids prepared by the enzymatic hydrolysis of casein was given intravenously as the only source of nourishment over an average period of 7 days.

The solution was found to be nontoxic. It could be administered quickly and easily. Although given intravenously at a rate of 30 grams of amino acid per hour it caused no significant reaction and appeared in the urine in insignificant amounts. In patients in whom excessive destruction of body tissue was not present evidences of plasma protein regeneration and of positive nitrogen balance were obtained.

REFERENCES

1. Addis, Thomas, Poo, L. J., and Lew, W. J. Biol. Chem., 1936, 115: 117.
2. Altshuler, S. S., Hensel, A. M., Sahyun, M. Am. J. M. Sc., 1940, 200: 239.
3. Cox, W. M., Jr., and Mueller, N. J. Proc. Soc. Exp. Biol., N. Y., 1939, 42: 658.
4. Daft, F. S., Robscheit-Robbins, F. S., and Whipple, G. H. J. Biol. Chem., 1938, 123: 87.
5. Elman, R. Ann. Surg., 1940, 112: 594.
6. Idem. Proc. Soc. Exp. Biol., N. Y., 1940, 43: 14.
7. Elman, Robert, and Weiner, D. O. J. Am. M. Ass., 1939, 112: 796.
8. Farr, L. E., and MacFayden, D. A. Proc. Soc. Exp. Biol., N. Y., 1939, 42: 444.

9. GOLDSCHMIDT, S., VARS, H. M., and RAVDIN, I. S. J. Clin. Invest., 1939, 18: 277.
10. HARTZELL, J. B., WINFIELD, J. M., and IRVIN, J. L. J. Am. M. Ass., 1941, 116: 669.
11. HOLMAN, R. L., MAHONEY, E. B., and WHIPPLE, G. H. J. Exp. M., 1934, 59: 269.
12. JOHNSON, J., RAVDIN, I. A., VARS, H. M., and ZINTEL, H. A. Arch. Surg., 1940, 40: 1104.
13. JONES, C. M., and ESTON, F. B. Arch. Surg., 1933, 27: 159.
14. MADDEN, S. C., and WHIPPLE, G. H. Physiol. Rev., 1940, 20: 194.
15. MADDEN, S. C., ZELDIS, L. J., HENGERER, A. D., MILLER, L. L., ROWE, A. P., TURNER, A. P., and WHIPPLE, G. H. J. Exp. M., 1941, 73: 727.
16. MECRAY, P. M., BARDEN, R. P., and RAVDIN, I. S. Surgery, 1937, 1: 53.
17. MUELLER, A. J., KEMMERER, K. S., COX, W. M., JR., and BARNES, S. T. J. Biol. Chem., 1940, 134: 573.
18. POMMERKENKE, W. T., GLAVIN, H. B., KARIHER, D. H., and WHIPPLE, G. H. J. Exp. M., 1935, 61: 283.
19. RAVDIN, I. S. Ann. Surg., 1940, 112: 576.
20. RHOADS, J. E., FLIEGELMAN, M. T., and PANZER, L. M. J. Am. M. Ass., 1942, 118: 21.
21. SHEARBURN, EDWIN W. Surg., Gyn. Obst., 1942, 74: 343.
22. SHOHL, A. T., BUTLER, A. M., BLACKFAN, K. D., and MACLACHLAN, E. J. Pediat., 1939, 15: 469.
23. THOMPSON, W. D., RAVDIN, I. S., RHOADS, J. E., and FRANK, I. L. Arch. Surg., 1938, 36: 509.
24. WEECH, A. A., GOETTSCH, E., and REEVES, E. B. J. Clin. Invest., 1933, 12: 217.
25. WHIPPLE, G. H. Am. J. M. Sc., 1942, 203: 477.

SURGICAL GLOVES AND WOUND INFECTIONS

LYLE A. WEED, Ph.D., M.D., and JESSIE L. GROVES, R.N.,
Indianapolis, Indiana

UNTIL about 1890 all surgical operations were performed with bare hands. Holmes and Semmelweis were among the first to report that the hands served to carry the organisms of puerperal sepsis, a fact which has been demonstrated over and over again. Since the work of Halsted and others in the 1890 decade, the use of rubber gloves has become an essential part of surgical technique as a means of preventing wound infection. Their use is taken as a matter of routine and, as in all matters which become routine, this step in the performance of a surgical procedure may become subject to a certain amount of unintentional carelessness. It is our purpose in this paper to emphasize that, although we realize that they are only one of several possible sources of infection, perforated gloves are an important source of infection in clean surgical wounds.

HISTORICAL

The infection of clean surgical wounds has been a major problem since the beginning of surgery. At first it was accepted as a normal risk of operation and the resulting suppuration was labeled "laudable pus." With the development of the germ theory of disease the presence of purulent exudate began to take on a new meaning. Surgeons began to realize that while phagocytosis was a normal phase of repair in infected wounds, healing was more rapid and the resulting scar was smaller if bacteria were eliminated from the wound. Since fermentation and putrefaction were brought about by bacteria present in the air, it was reasoned that this was the source of organisms infecting wounds. A wound was considered to be essentially an open culture tube with dead tissue as the substrate upon which bacteria could bring about fermentation and putrefaction. Since the air contained many bacteria capable of producing these changes and since the wound was exposed to the air, infection appeared to be a natural consequence. In order to prevent the development of such a condition, it was considered advisable to sterilize the air by spraying a suitable disinfectant into it in the vicinity of operation. A solution of phenol was used for this purpose in most operating rooms. However, by about 1895 surgeons were being impressed with the importance of the skin as a possible source of organisms. As early as 1891 Welch demonstrated that staphylococci occurred in stitch abscesses. The importance of the skin as a source of infection was again emphasized when Robb and Ghriskey in 1892 showed that it contained both staphylococci and streptococci as common inhabitants. These findings have been confirmed by many others.

Park recognized the fact that infection could and probably did come from several sources. Among the possibilities he listed as "the principal sources of contact infection: (1) skin and hair, (2) instruments, (3) sponges or their substitutes, (4) suture materials, (5) the hands of the surgeon and his assistants, (6) drainage materials, (7) dressing materials, (8) miscellaneous, e.g., drops of perspiration. . . ." He emphasized that the hands of all those concerned about the field of operation should be carefully disinfected. His concept of the importance of this procedure was vividly portrayed when he said, ". . . cemeteries have been filled in time past by the septic hands of medical students." Robb and Ghriskey isolated staphylococci and streptococci from the stitches and wounds of all the patients so examined. Their insight into the importance of the skin as a source of organisms is portrayed by the statement: "We have no sure and absolute method of rendering the field of operation entirely free from organisms, owing to the impracticability of destroying them in the superficial layers of the skin." No less an authority than McBurney stated in 1898: "My conclusion is that the real source of infection of a wound deliberately made by a careful surgeon who uses perfect materials and handles them perfectly is to be sought either in the skin of the patient or in the hands of those directly concerned in the operation." Also, "The writer does not claim that atmospheric dust is free from harmful germ life, but he does assert that, clinically, no evidence exists that such dust causes wound infection." Bovée commented in 1899 that "the air of the operating room is by no means free of pathogenic organisms, though this

From the Department of Bacteriology and the Training School for Nurses, Indiana University Medical Center, Indianapolis, Indiana.

Published with permission of the Research Committee, Indiana University Medical Center, Indianapolis, Indiana.

has probably been slightly exaggerated." He did not consider the introduction of rubber gloves alone sufficient to solve the problem, for he wrote: "We have cleansed our hands just as carefully to use the gloves as was usual without them. This is necessary as gloves may accidentally be perforated during operation." The use of cloth gloves was discredited by Lockett when he said, "The results of the above experiments seem to us to prove the absolute inefficiency of cotton gloves; both in their original state, and when paraffined they fail to prevent infection from the hands." While the use of gloves had already become routine in many places, leading surgeons continued to emphasize their importance after the turn of the century. Burford wrote: "that the operator's hands are the most dangerous sources of infection during operations is the most accepted view, at least in America. It seems conclusive that the operator's hands are more dangerous to the patient than the patient's own skin. . . . (p. 614). One having an infection on the fingers or the hand is not justified in operating while it lasts, even though the hands are covered by gloves, because of the risk of tearing the glove and leakage into the wound." Thomas considered gloves as having an indirect value to the surgeon when he facetiously remarked: "Gloves for unknown assistants are excellent, as the awe of the gloved hands prevents assistants from feeling impelled to feel the patient's pulse or open a door or window during the trying vicissitudes of any long operation." Brewer said that "by far the most important change in our technics which resulted from our bacteriological experiments was the adoption of the rubber gloves."

It was quite natural, therefore, that the interest should change from chemical sterilization of air to preoperative preparation of the hands of the surgeon and the skin of the patient. Many methods of skin sterilization were tried, but mercuric chloride appears to have been a favorite with a great many. The majority of such techniques, however, involved some combination of washing with soap, rinsing with ether or alcohol, washing with bichloride of mercury, and again rinsing with alcohol. Some operators also employed potassium permanganate and oxalic acid (12). In due time it was discovered that the chemical treatments employed were not as practical as had been believed, partly because the solutions were injurious to the hands, partly because they did not sterilize the surfaces in question.

In 1889 Halsted reported on the use of rubber gloves in surgery. Whether this was actually the first application of such a technique may be debated but a literature search has failed to establish any other priority. Within a few years many types of gloves were being advocated for use in surgery. Some were silk, some were cotton, and others were leather, some were rubber gloves without finger tips and some replaced the heavy finger tips with thin rubber finger cots. Because of the growing belief that passage of bacteria from the surgeon's hands to the wounds might take place through the cloth gloves, attempts were made to impregnate them with paraffin in xylol. Critical bacteriological tests, however, showed this did not make them entirely impervious (9). There was, therefore, a gradual adoption of rubber as the only safe operative glove to prevent wound infection.

By about 1900 rubber gloves were in common use in the better hospitals and private practice. However, there were many claims of greatly reduced incidence of infections by using cloth gloves (cotton or silk). Since Davidsohn (1888) had shown that instruments could be satisfactorily prepared for operative work by boiling, the method was automatically used for preparing the gloves, whether they were of rubber or cloth. Many surgeons, however, preferred chemical sterilization, usually with bichloride of mercury. Curiously enough, in a given operating team it was customary for only part of the individuals to wear gloves. In some cases the surgeons wore the gloves, in others the assistants wore them. Bloodgood in 1896 (3) has been credited with being the first to require all assistants to wear gloves, but the practice was not uniformly adopted until some years later. McBurney claims Halsted required his assistants to use them as early as 1891 but the writings of the latter in 1891–1892 do not mention it. Many objected to gloves as they interfered with the tactile discrimination considered the surgeon's best asset.

The adoption of the rubber glove as a standard part of good surgery was a very slow process. While some were reluctantly accepting them on their merits, others were recognizing certain inherent dangers in their use. As early as 1899 Bovée pointed out that even with the use of sterile rubber gloves it was still necessary to cleanse the hands just as carefully as without them. He said, "This is necessary, as gloves may accidentally be perforated during operation." He also emphasized that "the advantages of rubber gloves in surgery are, first, protection of patient against infection from operator's hands, and second, protection of operator's hands against infection from wounds."

Since the turn of the century, rubber gloves have gradually become an integral part of operative surgery in all reputable hospitals. The preoperative preparation of the hands has also continued as a part of the technique to lessen the hazard of infection from accidentally torn gloves as pointed out by Bovée. There has gradually developed the relatively uniform method of autoclave sterilization of the gloves in cloth packets. They are now usually wrapped individually and arranged for convenient aseptic manipulation by the washed hands of the one to wear them. Previously they were boiled in 5 per cent sodium bicarbonate solution. So long as the gloves remained intact, the chances of infecting a clean surgical wound were minimal. The importance of the glove today is certainly no less than in 1901 when Burford wrote: "Do our wounds heal better and more quickly with gloves than without? I can answer most emphatically that they have done so in my experience. With McBurney, I can say that I believe there is as much difference in the healing of wounds made with and without rubber gloves as there is between healing where the new method (antisepsis and asepsis) is and is not used."

The use of rubber gloves has not completely eliminated worry from infection of clean wounds. While many surgeons deny such developments occur in their practice, Meleney has pointed out that an unbiased critical analysis of the records will usually surprise the surgeon in charge. However, he was of the opinion the infections came directly from the respiratory tracts of the operating personnel. The extensive work of Hart has also emphasized the air as a source of infection in clean operative wounds. On the other hand, Devenish and Mills definitely traced a series of wound infections to one surgeon who was persistently perforating his gloves during operation. Still more recently Hirschfeld has emphasized the hands of the operator as a source of infection.

It thus appears that, as far as wound infection is concerned, scientific thinking has completed a 75 year cycle. During the time of Lister emphasis was placed on air as being a source of infection. Emphasis was then (1890–1910) changed to the skin of the patient and the hands of the surgeon. In 1898 Mikulicz commented, "The rôle of the air in operative infections has been greatly exaggerated since the beginning, especially by Lister." With the more recent work of Meleney and Hart, resulting in the development of ultraviolet irradiation, emphasis has again been placed on the air. During the past few years chemical sterilization of the air by means of aerosols has been rapidly advancing. It appears now there is a definite need for sterilization of the hands since rubber gloves are easily and unknowingly perforated during operation.

EXPERIMENTAL

During the course of some investigations on the bacterial content of the air in the operating room, recently reported by Rice and associates it was decided to determine the incidence of perforated gloves used in the Central Surgery of the Indiana University Medical Center.

In order to be fair to the surgical staff it should be emphasized that the data recorded herewith were obtained on gloves used by the entire personnel including residents in training, internes, student nurses, and other assistants common to the operating rooms of most university hospitals. It is the opinion of the staff members who have had extensive experience in other hospitals that the frequency of wound infection here is not greater than that found in other teaching hospitals.

The gloves were tested by distending with water and observing for leaks. A more accurate but less convenient method was to distend the fingers with air with the glove immersed in water and observe for bubbles. It is our impression this method is more certain to detect very small holes. However, because of lack of time on the part of the help responsible for examining the gloves, the examinations were made with water. A record was kept of the total number of gloves used during each operation, the number found perforated, the nature of the operation and the name of the surgeon in charge. Data were kept for each operation, irrespective of the type or importance or the number of gloves used.

The significant findings from data collected over approximately 20 months are given in Table I.

TABLE I.

Total number of operations (all kinds)	4,549
Number of operations with perforation of one glove or more	3,409
Per cent of operations with perforation of one glove or more	74.4
Total number of gloves used	35,763
Total number of gloves perforated	8,103
Per cent of gloves perforated	22.6

During the first 2 months the incidence of perforation was 32 per cent. There were two other separate months when the rate was higher than that given for the entire period, but these increases could not be traced to any individual. We

are not certain whether the reduced incidence after the first 2 months' observation is due to increased care on the part of the surgical staff or whether the coincidence is accidental. By the more accurate method of distending the gloves with air and immersing in water, we have examined 200 pairs of new unused gloves and have found none to be perforated.

We desired to obtain statistics on the incidence of wound infection among the patients operated upon in this surgery but consultation with the surgical staff emphasized the theoretical and practical difficulty in doing this. We believe no one factor can be used as a real index of minimal wound infection. Some patients develop a febrile reaction or leucocytosis or both without any grossly demonstrable evidence of wound infection. Others with obvious infection develop only a slight temperature or leucocytic reaction or both. To make cultures of the wounds would be meaningless since the surface of normal skin contains bacteria and the absence of growth on culture might mean either sterility or bacteriostasis due to the chemical used in preoperative preparation of the patient. It becomes a very difficult, if not impossible, task, therefore, to determine a minimal infection. Because of this no record was made concerning the incidence of wound infection during the period in which the gloves were examined.

The work of Price, Hirschfeld, and others have shown that many thousands of bacteria are present on the average hand after the usual routine preoperative preparation. We have confirmed these findings in all essential details (unpublished). Perforation of a glove during operation therefore becomes very important. If one hand contained only 10,000 bacteria uniformly distributed over the surface, 1 drop of perspiration in the finger tip could introduce a large number of bacteria. Furthermore, this is put directly into the wound when the perforated glove is on the hand of the surgeon. The practical importance of this is borne out by the work of Devenish and Mills.

SUMMARY

Data are presented showing the incidence of perforated gloves in the Central Surgery of the Indiana University Medical Center. The importance of perforated gloves as a possible source of infection of the clean surgical wound is emphasized. We believe some method of complete sterilization of the surface of the hands should be a part of modern surgery.

REFERENCES

1. Bovée, J. W. Am. J. Obst., 1899, 40: 491.
2. Brewer, G. E. Med. Rec., N. Y., 1898, 53: 433.
3. Burford, C. G. Northwest. Univ. Bull., 1901, 2: 607.
4. Davidsohn, H. Berl. klin. Wschr., 1888, 25: 697.
5. Devenish, E. A., and Mills, A. A. Lancet, 1939, 1: 1088.
6. Halsted, W. S. Johns Hopkins Hosp. Rep., Balt., 1890–1901, 2: 255.
7. Hart, D. Surgery, 1937, 1: 770.
8. Hirschfeld, J. W. Surg. Gyn. Obst., 1941, 73: 72.
9. Leedham-Green, C. Brit. M. J., 1896, 2: 1109.
10. Lockett, W. A. Philadelphia M. J., 1899, 3: 342.
11. McBurney, C. Ann. Surg., 1898, 28: 108.
12. Meleney, F. L. Surg. Gyn. Obst., 1935, 60: 264.
13. Mikulicz, J. Rev. chir., Par., 1898, 18: 936.
14. Park, R. Tr. Congr. Am. Physicians, 1891, 2: 29.
15. Price, P. B. J. Infect. Dis., 1938, 63: 301.
16. Rice, T. B., Weed, L. A., Raidt, H. Surg. Gyn. Obst., 1941, 73: 181.
17. Robb, H., and Ghriskey, A. A. Bull. Johns Hopkins Hosp., 1892, 21: 37.
18. Thomas, J. L. Brit. M. J., 1899, 1: 140.
19. Welch, W. H. Tr. Congr. Am. Physicians, 1891, 2: 1.

UNRELIABILITY OF BLOOD FINDINGS AS CRITERIA OF BURN SHOCK IN RABBITS

MILTON D. BOSSE, M.D., PAUL GROSS, M.D., and MARION L. HAGAN,
Pittsburgh, Pennsylvania

IT is generally accepted that hemoconcentration develops rapidly following extensive superficial burns of the skin. The degree and duration of the hemoconcentration have been considered very important prognostic signs of death or survival. The following experiments are presented because the results do not conform with those anticipated.

METHOD

Seventy rabbits, each weighing about 2 kilograms, were given extensive superficial burns of the skin while under ether anesthesia. The majority of the rabbits were New Zealand white males; the rest were of other breeds and a few were females. Nearly all of the animals were burned either by immersing the distal two-thirds of the body in water at 65 degrees C. for either 30 or 45 seconds, or by immersing the distal two-fifths of the body in water at 95 degrees C. for either 5 or 10 seconds. In most cases the area burned was thoroughly soaked in a 40 degree C. water bath immediately before and after burning. Ether anesthesia was of short duration and was only deep enough to prevent struggling. The animals were provided with amounts of food and water similar to that of normal stock rabbits. All preliminary red blood cell counts and other determinations on each animal were made on about 3.5 cubic centimeters of blood obtained from ear veins shortly before burning. Most of the determinations following the burn were made in each instance on about 3.5 cubic centimeters of blood obtained by cardiac puncture. Plasma proteins were determined by using the micro-Kjeldahl method for total nitrogen, subtracting the non-protein nitrogen, and multiplying the difference by 6.25. Rectal temperatures were recorded before and at intervals after burning. Hematocrit and clotting time determinations were also made. All surviving animals were sacrificed at intervals; up to 38 days following the burn. Autopsies were performed on practically all animals.

From the Western Pennsylvania Hospital Institute of Pathology, Ralph R. Mellon, M.D., director.
Presented at the St. Louis meeting of the American Association of Pathologists and Bacteriologists, April 3, 1942.

RESULTS

The mortality rate is shown in Figure 1. Fourteen animals, or 20 per cent, were dead 4 hours after the burn. Twenty-four more, or a total of 54 per cent, were dead within 24 hours. Twelve additional animals, or a total of 71 per cent, were dead within 3 days. Three other animals died 5, 7, and 10 days after the burn.

Differences in red blood cell counts after the burn compared to those before are expressed as per cent hemoconcentration in Figure 2. Wide variations in per cent hemoconcentration occurred at each period when red blood cell counts were made, but the majority of animals showed a mild to moderate increase at the 4 hour period after the burn. At 24 and 48 hours the red blood cell counts of the majority of animals had returned to normal, and at 72 hours the majority showed a decrease of 10 to 20 per cent below the normal.

Only 4 animals showed an increase in hemoconcentration of 40 per cent or more at the 4 hour period. One died within 24 hours, while the 3 others later showed a decrease in hemoconcentration. Two of these died during the next 24 hours, while the third survived.

Although 10 animals showed hemodilution at the 4 hour period, 8 of them died: 5 in 24 hours and 3 in 2, 5, and 10 days. The latter 3 showed progressive increase in hemodilution.

Forty-two animals had hemoconcentrations between 0 and plus 39 per cent at the 4 hour period. Eighteen were dead within 24 hours. The 24 others later showed either hemodilution or decrease in hemoconcentration, but 9 of these were dead within 3 days and another died 7 days after the burn.

Differences in plasma protein levels after the burns compared to those before are expressed as per cent change in blood proteins in Figure 3. Wide variations in per cent change in blood proteins occurred at each period when determinations were made, but at the 4 hour period the majority of animals showed a decrease of 31 to 50 per cent. Curves for the later periods are not drawn on the graph because the number of determinations was small, but there is a trend toward return of the protein level to normal or above.

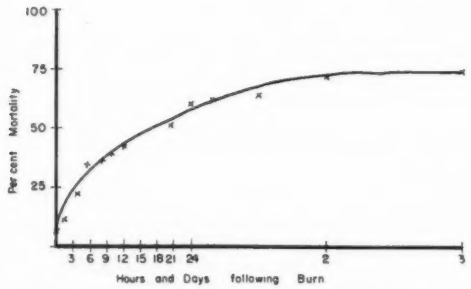


Fig. 1. Graph of mortality rate of rabbits following burn.

The 3 animals which showed little or no loss of plasma proteins at the 4 hour period nevertheless died within 24 hours after the burn.

One of the 9 animals that showed the most marked hypoproteinemia at the 4 hour period later showed a return to normal and 3 others showed improvement at the 24 hour period. However, all 4 died within 48 hours after the burn.

Seven rabbits developed plasma protein levels above the normal at the 48 or 72 hour periods. Three of these died within 3 days after the burn.

Immediately after the burn there was a rise of several degrees in rectal temperature, followed within a short time by a drop of 4 to 5 degrees C. below the control temperature taken just before burning the animal. The animals were depressed. Ear veins had collapsed within 10 minutes after the burn in practically all cases, so that these veins either did not bleed at all when cut, or bled very poorly. Most of the rabbits on which determinations were made showed a considerable rise of nonprotein nitrogen of the blood following the burns. Hematocrit readings in general were parallel to the red blood cell counts. The clotting time of the blood was usually shortened from the normal 4 minutes to 1 minute or less at the 4 hour period.

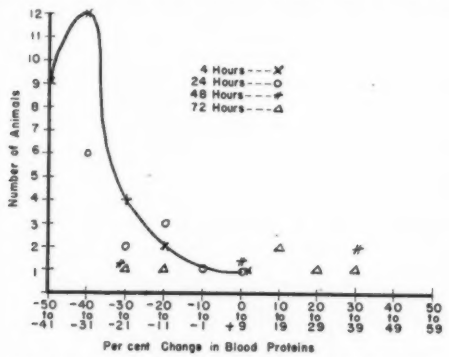


Fig. 3. Graph of changes in plasma following burn.

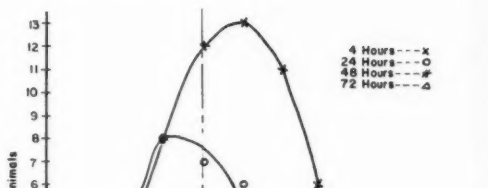


Fig. 2. Graph of hemoconcentration in rabbits following burn.

At autopsy, no significant pathological changes were seen in the viscera in most cases, regardless of the time of death after the burns. Two animals died of cardiac tamponade following cardiac puncture. Some rabbits showed petechiae of serous surfaces, mild or moderate pulmonary congestion or hemorrhage, and a few showed focal necrosis of the liver. Most of those that lived 12 hours or longer had a considerable accumulation of blood serum at the site of the burn.

OBSERVATIONS OF OTHERS

Underhill and associates found hemoconcentration occurring in most of their rabbits following light burns. The hemoconcentration was not maintained and in some animals it was quickly followed by dilution of the blood below the initial level. They also found some rabbits with hemodilution from the onset of shock. The tendency to hemodilution was explained by the hypothesis that the animals were able to draw upon the water reserves of the body to cause hemodilution either from the onset of the burn or following hemoconcentration. The animals in the experiments of Underhill and associates were admittedly only lightly burned over a relatively small area and the assumption was made that if the burns had been of sufficient gravity, the animals would have been unable to compensate. Our experiments do not support this assumption inasmuch as hemodilution occurred in a significant number of fatally burned rabbits.

Moon (1) believes that the response of rabbits to shock-producing conditions such as burns is variable. The present theory of the mechanism

of shock as described by Moon (2) is supposed to apply to all animals which exhibit this phenomenon. The unreliability of blood findings as criteria of shock in the rabbit would appear to throw some doubt on the applicability of the theory to this animal.

SUMMARY

Seventy rabbits were burned by partial immersion in hot water while under ether anesthesia. Although the mortality rate was high, the majority of animals showed only mild or moderate hemoconcentration and the degree or duration of hemoconcentration were not accurate indications of the prognosis for survival or death. It was found in a number of rabbits which died that the hemoconcentration was quickly followed by either hemodilution or decrease in hemoconcentration and in several there was hemodilution from the onset. Hypoproteinemia was more marked and occurred in most of the animals on which determinations were made, but some discrepancies were also encountered. Several animals with little or no hypoproteinemia died and several others, with earlier low plasma protein levels which subsequently improved or in some instances even returned to normal, also died. These results do not conform with those which have been reported on other animals.

REFERENCES

1. Moon, V. H. Discussion at St. Louis Meeting of the American Association of Pathologists and Bacteriologists, April 3, 1942.
2. Idem. Shock and Related Capillary Phenomena. New York: Oxford University Press. 1938.
3. Underhill, F. P., Kapsinow, R., and Fisk, M. E. Am. J. Physiol., 1930, 95: 302–314.

EDITORIALS

SURGERY
Gynecology and Obstetrics

FRANKLIN H. MARTIN
Founder and Managing Editor
1905–1935

LOYAL DAVIS, EDITOR

Associates
SUMNER L. KOCH MICHAEL L. MASON

DONALD C. BALFOUR, *Associate, Editorial Staff*

NOVEMBER, 1942

"GAS PAINS?"

THE early recognition of ileus, whether occlusive or nonocclusive in type has long been recognized as being of importance in control of mortality from this condition. Treatment whether by early operation, intestinal intubation plus operation, or intubation alone, is easier and more efficacious if begun early. The phenomena associated with persistent or marked distention frequently may be averted or at least minimized by early treatment.

Text material concerning ileus lists as cardinal signs, abdominal distention and obstipation. It is well to point out that these are not early signs of bowel obstruction. When distention is sufficiently marked as to be visibly or palpably obvious, the situation is no longer early as changes subsequent to distention have already taken place. Except in very thin persons distention of marked degree may be present without being obvious.

It is not unusual to hear the thought expressed that because a patient passes gas or has a bowel movement, ileus is not possibly present. It is not rare for patients with bowel obstruction to have some diarrhea, and passage of gas or feces is not uncommon. Partial obstruction, obstruction high in the gut, or segmental inactivity with hyperactivity in the lower bowel accounts for the passage of gas and feces long after the bowel is obstructed.

Diagnosis of ileus is made early by those alert to the possibility of its presence. Persistence of so called "gas pains" ought not be necessary to suggest ileus. The term "physiological ileus" ought not be allowed to cloud the fact that gas pains are not necessary in the majority of cases. Their presence is not due alone to the quiet imposed upon the intestine by the trauma of the operation but also by excessive gas in a reacting gut.

It has become customary, indeed thought almost necessary, for a vast array of laboratory studies to accompany a diagnosis of ileus. The expense of such studies, which are not likely to yield information of value in diagnosis, is frequently not considered. There is but one laboratory finding of importance in the diagnosis of ileus and that consists of a flat film of the abdomen. This is a simple, painless laboratory procedure which yields rapid and definite data regarding the presence of gas in the small bowel long before abdominal distention is evident. The basic cost to the hospital of such study is less than that usually charged for ordinary blood chemical study and accordingly, the use of films of the abdomen, by portable machine if necessary, ought be encouraged.

There is still fear, apprehension, and distaste in some remote corners of medical practice to the passage of a tube into the stomach.

duodenum, or intestine of a postoperative patient. All too commonly the concept toward postoperative vomiting resembles that of the Duchess in *Alice in Wonderland* who says "speak harshly to your little boy, and beat him when he sneezes; he only does it to annoy, because he knows it teases." The patient who vomits usually does so to empty the stomach. Restriction of fluids and a lecture is not good treatment for postoperative gastric distention or vomiting. Aspiration of the stomach contents through a nasal catheter is much more pleasant and efficacious. Vomiting is hard, unpleasant work and one "upheaval" will more than equal the hardship attendant upon the passage of a tube. There is no excuse for persistent postoperative vomiting, and he who permits it courts not only the displeasure of a tired, distressed sick patient but the likelihood of distention and dehydration with serious and possibly lethal results.

CHARLES G. JOHNSTON.

UNRECOGNIZED OBSTRUCTION AT THE VESICAL NECK

"AND Moses was an hundred and twenty years old when he died: his eye was not dim nor his natural force abated."[1] Brim[2] states that a correct literal translation of the second part of this passage from the Hebrew scripture should read "his secretions did not escape." Thus, the writer of the ancient scripture in a boasting manner declared to the world that the physical status of the aged patriarch was so nearly perfect that unlike other old men of his times Moses suffered no loss of vision from senile cataracts and did not dribble urine. The antiquity of the problem of urinary retention is, therefore, unquestionable according to Brim.

[1] Deuteronomy 34:7 (King James translation).
[2] Brim, C. J. Medicine in the Bible. New York: Froben Press, 1936, p. 352.

It is difficult to comprehend how it was possible for several thousand years to elapse without the appearance of even a partially successful solution of the problem of urinary retention, whereas within the space of one generation, even within the memory of men still living, an almost complete solution of the difficulty has been accomplished. The turn of the century witnessed much controversy and keen discussion among surgeons of that day as to the proper method of removal of the enlarged prostate gland for the purpose of relief of retention of urine. Shortly thereafter, the surgical procedures of perineal and suprapubic prostatectomy became well established. Although the transurethral method of operation on the prostate gland was among the first to be tried, it was not until the past decade that it was developed to such a point that it is now a standard surgical procedure, applicable in routine urological practice.

Of the present status of the subject of urinary retention in the male, this much can be said: in most cases obstruction at the neck of the bladder is readily recognized and the pathological condition is efficiently corrected by surgical treatment. Digital palpation of the prostate gland through the rectum and cystoscopic examination yield information which is sufficient in most cases to permit the making of an unqualified diagnosis of obstruction of the vesical neck caused by enlargement of the prostate gland or fibrotic contraction of the vesical neck.

If a casual survey is made of the modern problem of urinary retention, the pleasant picture is obtained of a large number of grateful patients well cared for. Closer examination, however, brings to attention the fact that there is also a small group of hapless patients suffering with urinary retention who still wander from physician to physician, seeking but not finding relief from their malady. These

are cases in which it is difficult or impossible for the physician to demonstrate any definite obstructing lesion, by means of palpation, cystoscopy or open surgical exploration. When it is impossible to demonstrate an obstructing lesion, it is only natural that the profession should grope for reasons to explain the retention. "Atony" and "hypotonicity" of the detrusor urinae muscles as a result of "congenital factors" or "cord" or "neurogenic" bladder are vague terms which have been employed in such cases in spite of the fact that lesions of the central nervous system sufficient to cause such disturbances could not be clearly demonstrated. Most unjustly maligned as the ogres of this unhappy state have been syphilis of the central nervous system and its stepchild, tabes dorsalis.

There are many scientific facts that cannot be demonstrated by the five senses, but must be ascertained by the process of deduction. Recognition of this truth has finally brought light to the obscure condition of urinary retention of undetermined etiology. The realization that obstruction may exist, even though it is not readily demonstrable, has brought relief to many suffering patients. It is becoming widely recognized that minimal degrees of obstruction, such as fibrosis, mild contraction of the vesical neck, and thin collars of adenomatous tissue surrounding the vesical neck, which may defy the detection of even the most experienced urologist, can be the source of the retention. Apparently, an almost normal vesical neck which has lost some of its resiliency can be the offending lesion. This is more likely to be true when it is associated with a bladder weakened as a result of interference with its sensory nerve supply, as is occasionally seen in the presence of tabes dorsalis and allied conditions.

This particular phase of the problem of urinary retention has been late in solution, because of the fact that until the last decade no satisfactory method of removal of minimal amounts of obstructing tissue (and, at the same time, preservation of a smooth, conical shaped prostatic urethra with no postoperative deformity) has been available. By means of transurethral operation this ideal finally is possible. Every experienced surgeon in this field now realizes that it is unwise to make the statement that no obstruction of the vesical neck exists until after transurethral resection actually has been attempted. In many cases, as such resection proceeds, obstructing tissue that was not recognized previously becomes apparent; almost normal-appearing vesical necks at times can be widened to several times their previous diameters. Bladders weakened and stretched because of long years of urinary retention again respond to the stimulus of micturition and in most cases eventually completely empty their contents. The myth of the "atonic bladder" is about to be exposed, and the group of patients who have urinary retention of "undetermined etiology" is rapidly diminishing in size.

Of interest in the future will be the gradual solution of many of the difficulties of micturition which result from genuine and demonstrable lesions of the central nervous system. Already, interesting results are being obtained from transurethral surgical procedures on the vesical neck, and in some cases results are accruing which may radically alter our opinions concerning the physiology of micturition. The physician interested in this subject will watch with interest the reports of such work as they are published from time to time in the medical literature.

JOHN L. EMMETT.

THE SURGEON'S LIBRARY

REVIEWS OF NEW BOOKS

THE excellent monograph on *The Treatment of Burns*[1] by Harkins is published at a most appropriate time, as the subject is one of importance to both the industrial and military surgeon. It has brought up to date the world's literature on the subject of treatment of burns.

In offering a word of criticism one wonders if the younger surgeon or student, after reading the volume, would be able to plan and carry out an effective method of treating a burned patient because so many different methods are described as almost to cause confusion.

One serious omission, in the reviewer's judgment, is the lack of emphasis, almost of mention, of the importance of primary cleansing of the large open wound and of preventing subsequent infection. If we have not yet become aware of the importance of masking the patient and the personnel during treatment, early and late, of large open wounds, then we shall continue to have serious infections of patients with burns. The evidence regarding infection has been well presented in Chapters 9 and 10 but the obvious conclusions are omitted.

In the chapters on "General and Local Care" of the burns the question of cleansing of the open wound is only briefly suggested by reference to the "Tschmarke method" under the heading of débridement. Certainly the beginner in surgery, after reading these chapters, would fail to realize that conversion of the contaminated wound into a clean wound is a primary principle in the treatment of a burned surface.

HARVEY ALLEN.

THE valuable and practical book *Métodos Modernos de Amputação*[2] is one of the best presentations of amputations we have seen. The material is based on the author's large experience, study of postmortem material, and thoughtful consideration of some five hundred articles in the surgical literature of South and North America, England, and continental Europe.

The first and second chapters deal with general considerations, including discussions of optimal levels of amputations and technique. In the upper extremity the optimal levels are shown to be different in the intellectual man and in the laboring man. The principles of dealing with the skin, aponeurosis, muscle, bone, cartilage, synovia, nerves, and vessels are fully presented. The third chapter discusses the indications for amputation.

The fourth and fifth chapters are taken up with detailed descriptions of individual amputations. This section is made vivid and instructive by a wealth of splendid drawings by José Gonçalves Filho. For teaching value, these drawings far exceed photographs or simple line diagrams, and they deserve highest commendation.

The final chapter is a practical and well illustrated description of the various prostheses suitable for different types of amputations.

FREDERICK CHRISTOPHER.

A VERY definite need of a practical book dealing with less extensive surgical procedures is filled by Ferguson's *Surgery of the Ambulatory Patient*.[3] The teaching of surgery in most medical schools is concentrated on major hospital procedures and often too little attention is paid to the care of so called minor surgery. This lack is exhibited by the confusion of most internes when they begin their services in hospital out-patient departments. As is emphasized by the author, surgical procedures which can be performed in a physician's office or hospital dispensary are very numerous. Many of them require as much diagnostic skill, surgical ability and judgment, and meticulous care as major hospital procedures. Poorly done, they may produce lasting disabilities. Many surgeons prefer to confine all of their operative work to hospital patients and will hospitalize some who could as well be handled in the office. This has some advantage for the surgeon but the added expense and loss of time may be an unnecessary burden to the patient.

The extensive experience of the author in dealing with surgical lesions of ambulatory patients has made him familiar with this field. He has gone into detail to supply all necessary information for the satisfactory conduct of office surgery. Office equipment and preparation is described minutely. The advantages of various forms of anesthesia and methods of administration are clearly outlined and illustrated. Preoperative preparation, dressings and bandages, and subsequent care are explained. The book is divided into three main sections: one describing the nature and care of typical lesions; the second, devoted to regional surgery; and the third, to fractures and dislocations. Many contributions have been made to the book by associates of the author which have added authoritative information

[1] THE TREATMENT OF BURNS. By Henry N. Harkins, M.S., M.D., Ph.D., F.A.C.S. Springfield, Ill., and Baltimore, Md.: Charles C. Thomas, 1942.

[2] MÉTODOS MODERNOS DE AMPUTAÇÃO. By Edmundo Vasconcelos. São Paulo: Companhia Editora Nacional, 1942.

[3] SURGERY OF THE AMBULATORY PATIENT. By L. Kraeer Ferguson, A.B., M.D., F.A.C.S. Philadelphia, London, and Montreal: J. B. Lippincott Co., 1942.

in special fields. Anatomical drawings of the body and skeleton with page listings of lesions of each part make a very practical index.

In general, I think the book an excellent and practical guide for office and dispensary surgery. Perhaps more emphasis could be placed upon the dangers of surgery of patients not hospitalized. Many procedures are included which are quite major and which should be done on ambulatory patients only under most favorable conditions and when hospitalization is impracticable.

CHARLES B. PUESTOW.

THE basic organization of a medical department is considered in detail in Lasher's *Industrial Surgery*[1] which is a well written book based on an extensive industrial practice. The book should be of great value to the host of small industries in which little is known of a medical department and how to go about organizing it.

The present national emergency has brought out the fact that the industrial surgeon is by far the most valuable individual at home to care for the workman and in the field to care for our fighting forces. It is foretold that in the very near future large universities will establish a special department dealing with industrial medicine and surgery to give a previously ignored group adequate skilled training.

One hundred and sixty-eight infections among 20,919 fresh wounds is a rather high number. The high percentage of infections ties in well with the statement of policy expressed on page 174 that "small compound wounds are swabbed with tincture of iodine, and covered with sterile gauze and left undisturbed *UNLESS . . .*"

The experience of the author and the analysis of malingering is well dealt with. Fractures as a whole are well handled. In injuries to the hip, the author is of the opinion that the Whitman closed reduction compares favorably with those obtained by other methods.

The recognition that dental services be rendered by the industrial medical department is to be highly commended in the form of educational activities, diagnostic service, prophylactic care, emergency treatment, and operative dentistry limited to simple extractions and fillings.

The subject of electrical shock is handled especially well when the author states that as long as eight hours may be required before the paralyzed respiratory center responds to treatment.

The approach to the femoral hernia through an inguinal incision is to be commended.

The author believes that frankness, impartiality, frequent consultations and repeated x-rays (often shown to the patients and explained to them) will go far in gaining the whole-hearted co-operation of the workman.

R. J. BENNETT.

AN attempt is made in Schauffler's *Pediatric Gynecology*[2] to combine into one small volume a list of subjects from the embryology of the female pelvis to the medicolegal aspects involved after the pelvic organs have performed their mature function. The management and psychological approach to the child plus a description of the preferred technique in examining small apprehensive patients is stressed throughout the volume. It is a combination of good common sense plus a keen insight into the mental reactions of children. The discussion in places is a bit vague and not as descriptive as the subject justifies.

Common but distressing problems, such as diaper rash, are given adequate treatment. Many practical points are covered under the section on vaginitis: the proper technique in taking a vaginal smear and its inadequacy in making a diagnosis, the value of cultural methods in the diagnosis of gonorrheal vaginitis, the insistence that acutely ill children with incidental gonorrheal can be safely admitted to general hospitals if proper isolation technique is carried out, the condemnation of child-caring institutions, and the recommendation of foster homes for the care of gonorrheal vaginitis. These numerous points are concluded with a description of the simplified treatment for gonorrheal vaginitis, suggesting that, at the present time, sulfathiazole is the drug of choice.

In the section on hormone therapy, the author sounds a much needed note of warning against their promiscuous use at a very unstable period in a child's life. The usual and many times unsuccessful treatment of enuresis is covered; also the questionable procedure of trying to train an infant to toilet habits before it is able to sit up is advocated. A rather large section is devoted to the social service medicolegal problems that arise in dealing with this subject. This book will be of value in making one consider the gynecological problems of children in their entirety.

L. MARTIN HARDY.

THE 6th edition of one of the most popular British textbooks of medicine[3] has recently been released. The book is written on the multiple author plan by an unusually well chosen and eminent group of English authors. The editor has had the foresight to limit the number of contributors so that for the most part each of the 21 sections has been written by a single individual. Thus many of the objections to a multiple author textbook of medicine are overcome and yet most of the advantages are maintained.

The text is in one volume and includes sections on dermatology, tropical medicine, and psychological medicine. In spite of the 2000 odd pages it is only slightly thicker than most one volume medical textbooks, thanks to especially thin paper.

[1] INDUSTRIAL SURGERY; PRINCIPLES, PROBLEMS, AND PRACTICE. By Willis E. Lasher, M.D., F.A.C.S. New York and London: Paul B. Hoeber, Inc., 1942.

[2] PEDIATRIC GYNECOLOGY. By Goodrich C. Schauffler, A.B., M.D. Chicago: The Year Book Publishers, Inc., 1942.

[3] A TEXTBOOK OF THE PRACTICE OF MEDICINE. By various authors. Edited by Frederick W. Price, M.D., C.M., F.R.C.P., F.R.S. (Edin.). 6th ed. London: Oxford University Press, 1941.

The subject matter is treated in a conventional manner with emphasis on those things of a practical nature. The discussion of etiology and pathology and theoretical material is rather brief. In this edition the text has been brought entirely up to date and thoroughly revised. In the reviewer's opinion this is one of the best single volume texts of medicine available. RICHARD B. CAPPS.

THE history of and indication and technique for the operation of vaginal hysterectomy are dealt with in Kennedy and Campbell's *Vaginal Hysterectomy*.[1] The historical data are very complete and bring the reader up to the present time. The indications for vaginal hysterectomy and the surgical technique are exhaustively set forth; and the advantages of the vaginal operation are contrasted with those of the abdominal procedure in an attempt to evaluate the merits of each operation in certain types of cases.

The vaginal hysterectomy operation is contrasted with the use of x-ray and radium in treatment of malignant disease of the uterus. The use of the cautery in connection with vaginal hysterectomy is discussed.

Considerable space is given to the interrelationship between the ovary and the uterus as regards hormonal stimulation, and the bearing this subject has on hysterectomy is freely discussed.

The postoperative complications which may develop are clearly set forth and discussed, and postoperative treatment is outlined.

Dr. Kennedy presents a series of illustrations showing in detail vaginal hysterectomy by the clamp method. Each illustration has an exhaustive legend and sets forth in detail the steps of the operation shown in the accompanying full page drawing. The pathology of the various conditions which indicate vaginal hysterectomy as well as the steps in the operative procedure are illustrated. Associated pathological conditions, such as cystocele and rectocele, are also discussed and the steps in the operation for their cure are carefully outlined.

Dr. Campbell in Part 3 takes up the technique of vaginal hysterectomy by the ligature method. He describes at length the factors entering into the production of uterine procidentia, laying special emphasis on the anatomy. Preoperative and postoperative care together with indications for the operation are thoroughly discussed. Complications immediate and remote are also described, and appropriate suggestions are offered for the care of complications which may occur. This section is supplemented by a series of 21 halftone illustrations which depict removal of the uterus by the ligature method and many important associated anatomical relationships between the genitalia and other pelvic and abdominal structures. Many important points in diagnosis and technique are shown and stressed by the author.

This book is well written by two masters of the technique which they have described. It clearly sets forth the advantages of each method and leaves the reader free to make his choice of operation. Many valuable suggestions may be gleaned from careful reading of this treatise by the inexperienced surgeon as well as by men who are familiar with general surgery, and also those who have had considerable experience in this particular field. The type is exceptionally clear and readable and the paper is excellent. At a time when vaginal hysterectomy is enjoying more and more popularity, it is a book that every surgeon can well afford to read and digest to help improve his technique in respect to this operation. F. H. FALLS.

IN an interestingly written book of 450 pages the author of *Surgical Physiology*[2] briefly summarizes the more important aspects of physiology as they concern the surgeon. The organ systems are each covered in separate chapters. The material covered is hardly extensive enough for a reference work as the author did not intend that it should serve such a purpose. For this reason, however, it is open to the criticism of all such concise reviews in that some sacrifice of accuracy and completeness must be made. The recent literature is fairly adequately if slightly uncritically reviewed.

Collecting such an amount of material into a single volume was a tremendous task. The production of a readable and stimulating book on a subject with as extensive a literature as physiology as it applies to surgery is a real contribution, and the author is to be congratulated.

THOMAS C. DOUGLASS.

THE book *Pathology of the Oral Cavity*[3] by Lester R. Cahn is a well prepared treatise on the common pathological conditions of the oral cavity, with excellent histopathological illustrations. Many good clinical photographs and x-ray films add to the practical value of the text, although clinical diagnosis and treatment are generally omitted.

The chapter on cysts and tumors of the jaw bones and on the histology and pathology of the soft tissues are very well done. The chapter on diseases of the hard structure of the teeth, including dental caries, might well have been omitted, as well as that on pyorrhea. As a concise and pertinent text on histopathology of oral lesions, it is an addition to the literature and one which is conveniently arranged for practical use. CHARLES W. FREEMAN.

[1] VAGINAL HYSTERECTOMY. By James William Kennedy, M.D., F.A.C.S., and Archibald Donald Campbell, M.D., C.M., F.R.C.S.(C), F.R.C.O.G., F.A.C.S. Philadelphia: F. A. Davis Co., 1942.

[2] SURGICAL PHYSIOLOGY. By Joseph Nash, M.D. Springfield, Ill., and Baltimore, Md.: Charles C. Thomas, 1942.

[3] PATHOLOGY OF THE ORAL CAVITY. By Lester Richard Cahn, D.D.S Baltimore: Williams & Wilkins Co., 1941.

AMERICAN COLLEGE OF SURGEONS

1942 CLINICAL CONGRESS CANCELLED

THE annual Clinical Congress of the American College of Surgeons, which was scheduled to be held in Cleveland November 17 to 20, 1942, was cancelled by the Board of Regents of the College at a meeting held in Chicago, Wednesday, October 14. Motivated primarily by patriotism, the regents were influenced by the present conditions surrounding the general war program which have led to a greater burden on the members of the surgical profession in their local communities as a result of the large proportion of the profession which is serving with the armed forces. The regents by this action took cognizance of the desire of the profession to do nothing which would interfere with the successful prosecution of the war program such as would be caused by temporary absence of its members from civilian duties during the period of the Congress, embarrassment of the transportation system, and interference with the work of the local profession in Cleveland in preparations and presentations incident to such a meeting.

At the annual meeting of the Board of Regents, which will be held in December, fellowship in the College will be conferred in absentia on the class of initiates of 1942 as there will be no convocation exercises. At the same time the list of hospitals, cancer clinics, medical services in industry, hospitals conducting programs of graduate training in surgery, and medical motion pictures, that meet the College standards will be approved and later published.

All present officers, governors, regents, and standing committees will continue in office.

of the
ortation
he local
nd pres-

Regents,
rship in
on the
no con-
e list of
s in in-
raduate
pictures,
pproved

nts, and
ce.